# EFFECTS OF DIFFERENT METHODS, SOURCES AND LEVELS OF SELENIUM SUPPLEMENTATION AND FERTILIZATION ON BEEF CATTLE AND FORAGE TISSUE LEVELS

BY

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Dedicated to:

My wife Alba, for her love, support and understanding in my profession,

My children, Michelle, Claudia and Guillermo, for being three bright lights in my life,

My mother and father, for their teachings, love and support,

My brothers Jorge Luis and Marco Antonio, and especially my sister, Lourdes.

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Abstract of Dissertation Presented to the Graduate School
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# EFFECTS OF DIFFERENT METHODS, SOURCES AND LEVELS OF SELENIUM SUPPLEMENTATION AND FERTILIZATION ON BEEF CATTLE AND FORAGE TISSUE LEVELS

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Several experiments were conducted in Northwest and Northcentral Florida to determine effects of different methods and sources of selenium supplementation and fertilization of forages on the performance and tissue levels in beef cattle and on tissue selenium levels of two forage species. Organic selenium (Se-yeast) proved to be more effective in stimulating weight gains and liver selenium concentrations than an inorganic source (sodium selenite) for Angus steers and heifers. Plasma selenium was also higher with the organic source, although none of the sources achieved adequate levels in the plasma. However, in Angus steers and heifers neither the organic nor the inorganic selenium sources were able to alter plasma concentrations of the thyroid hormones $T_3$

and $T_4$ after administration of thyrotropin-releasing-hormone. In adult Angus cows, a selenite (Mu-Se) treatment was not effective in raising plasma selenium concentrations while barium selenate (Deposel) maintained a constant level in plasma. Neither the Mu-Se or Deposel treatments attained plasma selenium adequacy (0.07 mg/L) but were above the critical level (0.03 mg/L). The organic (Se-yeast) mineral mixture treatments did increase plasma selenium concentrations and attained adequacy. The control and Mu-Se treatments did not achieve adequate selenium concentration in liver at any time, but the Deposel did so at 24 mo. The salt mineral mixtures (Se-yeast) were adequate at 6, 12, and 24 mo. Concentrations of selenium in milk were not adequate and declined with time as did the colostrum selenium concentrations. Neither dam injected Mu-Se or Deposel was effective in raising or maintaining calf plasma selenium concentrations and the trend was to decrease with time. However, the free-choice mineral mixtures (Se-yeast) were effective in attaining and maintaining adequate plasma selenium concentrations in calves. Spraying of Selcote-Ultra, a selenium fertilizer, on bahiagrass was not very effective in raising forage selenium to adequate levels (>0.1 mg/kg) beyond 8 wks at the 5 g/ha rate while the 10, 15, and 20 g/ha rates were still adequate at 12 wks after application. Topdressing of Selcote-Ultra on fescue is an adequate method to increase forage selenium as the levels were more than adequate 22 wks after application. Organic selenium (Se-yeast) was more effective in raising and maintaining adequate selenium concentrations in tissues of beef cattle while the fertilizer worked better when topdressed on forage than when sprayed.

# CHAPTER 1
# INTRODUCTION

Selenium is an element that was first considered toxic for livestock. However, the role of selenium as a nutrient was first demonstrated by Schwarz and Foltz (1957), who found that it was a component of "factor 3," which prevented liver necrosis in rats. The role of selenium as an essential micronutrient is well characterized for domestic animals (Oldfield, 1987) and evidence is accumulating indicating its health protective effects in humans (Milner, 1995). Selenium deficiency impacts on cattle health include nutritional myodegeneration (NMD), retained placenta, infertility, abortion, birth of premature or dead calves, cystic ovaries, metritis, delayed conception, erratic and/or silent heat periods, poor fertilization rates, diarrhea, decreased immune response and "ill thrift" (Maas, 1983; Corah, 1991; Gerloff, 1992; Hansen and Deguchi, 1996).

Selenium is involved in cellular antioxidant systems, mainly via peroxidase enzymes, thyroxine metabolism, and arachidonic acid metabolism (Neve, 1991). Selenium accumulates in the mammalian testis to a greater extent than it does in other tissues and enhances the motility of spermatozoa (Behne et al, 1986). Thus, it can be assumed that, besides its well known effects on the oxidative defense system through its function in glutathione peroxidase, it may play an essential role in the fertility of animals and man.

Selenium deficiency in animals, when fed rations and/or feed crops containing less than 0.1 mg/kg selenium, occurs in many countries (Gupta and Watkinson, 1985; Kivisaari, 1998). In North America, selenium deficiency occurs widely, e.g., in the Northeast, Southeast, in the Pacific Northwest, regions lying east of the Mississippi river and in areas where sulfur-based fertilizers depress plant selenium uptake (Welner, 1996; Gupta and Gupta, 2000). During the last decade selenium deficiency in livestock has been recognized as a problem in many areas of the world. As a consequence, addition of selenium to feedstuffs has been widely used to provide an adequate supply (Hansen and Deguchi, 1996). Supplementation of dairy cattle diets with 0.3 mg/kg DM selenium usually maintains plasma concentrations of selenium within normal ranges (Maus et al., 1980) and is generally considered adequate (NRC, 1988).

However, controversy still exists on how much selenium to supplement, the use of inorganic or organic sources, and at what level toxicity occurs, etc. Recently, Ellis et al. (1997) concluded that dietary intakes of selenium as (sodium selenite) as high as 50 to 100 mg/d can be tolerated by dairy cattle for several wk without adverse clinical consequences. The dosage of sodium selenite necessary to cause chronic toxicosis in cattle appears to be in the range of 0.25 to 0.50 mg/kg body weight over a period of several weeks. Retention, bioavailability, storage potential, absorption and toxicity also differ between organic and inorganic selenium.

With the above considerations in mind, several experiments were conducted in Northwest and Northcentral Florida to determine the best method of selenium supplementation, the best source and level of selenium for the supplementation, its effects on thyroid metabolism and on growth of calves, and the best way to administer a new selenium fertilizer for two different forage grasses.

# CHAPTER 2
# LITERATURE REVIEW

## The Role and Importance of Selenium in Animal Nutrition

The essentiality of selenium as a nutrient was first demonstrated by Schwarz and Foltz (1957), who found that it was a component of "Factor 3," which prevented liver necrosis in rats. Other diseases were soon shown to be a consequence of selenium deficiency in animals, including skeletal and cardiac myopathies (Combs and Combs, 1986). Impacts on cattle health have been associated with a selenium deficiency and may include nutritional myodegeneration, retained placenta, infertility; abortions, premature births, weak or dead calves, cystic ovaries, metritis, delayed conception erratic, weak or silent heat periods, poor fertilization rates, diarrhea, decreased immune response and "ill thrift" (Maas, 1983; Corah, 1991). Selenium seems to be essential for normal spermatozoa development in both experimental animals and in livestock. (Hansen and Deguchi, 1996). Orally administered selenium is effective in reducing incidence of infertility in ewes and in an increasing number of born lambs. It also increases uterine contractions thereby increasing the chance of fertilization (Hansen and Deguchi, 1996).

Selenium has a very high metabolic rate that allows it to function at low dietary levels, but this property can make it a "double edged sword," meaning that at higher levels it becomes toxic. Toxic levels are about 100 times the required levels (Oldfield, 1998). There is still much debate on what the required level is and on what level would be toxic. An amendment to the selenium regulation was approved in 1978 indicating that supplemental selenium could be used up to 0.1 mg/kg in domestic animal diets (FDA, 1978). Later, approval was granted for an increase in the maximum level of supplemental selenium from 0.1 to 0.3 mg/kg in the feeds of the major food producing animals (FDA, 1987a,b).

Support for this higher level has been found by several investigators (Smith and Conrad,1987; Stowe et al., 1988; Podoll et al., 1992) who suggested that selenium intakes sufficient to support adequate serum concentrations in lactating dairy cows should be 5 to 7 mg/d. A lactating cow consuming 20 kg of dry matter/d (with 0.3 mg/kg selenium) would have a daily intake of 6 mg, in addition to any selenium in the forage. According to Hakkarainen (1993), the limit for chronic toxicity in diet is about 3-5 mg/kg and not at 10 mg/kg , as previously believed. Negative physiological effects have been reported even at levels around 1 mg/kg (Larsen et al., 1988; Larsen and Tollersrud, 1981; Moksnes, 1986; Spallholz et al., 1973a,b). Such negative effects include a decreased resistance to disease, poorer growth, and other signs. Particular care must be taken with respect to the over supplementation of selenium to growing and pregnant mammals because, according to Thorlacius-Ussing (1990), selenium occurs in rather high concentrations in the anterior pituitary of the rat and human and an overdosage of selenite results in a long-lasting toxic effect on the growth performance of young rats. Different maximum tolerable levels have been proposed for selenium, ranging from 2 mg/kg in the diet (NRC, 1996) to 4 or 5 mg/kg (Underwood, 1977). With 5 mg/kg of selenium in diet, signs of toxicity take weeks or even months, to appear (Underwood, 1977).

The minimum lethal dosage of inorganic selenium required to induce acute toxicosis is in the range of 3 to 5 mg/kg of body weight for most species, but may be twice that for cattle (Ellis et al., 1997). The dosage of selenium as sodium selenite necessary to cause chronic toxicosis in cattle appears to be in the range of 0.25 to 0.5 mg/kg of body weight over a period of several weeks. Ellis et al. (1997) did not find outward signs of selenium intoxication in cows in which the maximal daily selenium dosage was 0.087 mg/kg for 90 days or 0.174 mg/kg for 28 days. In animals with selenium toxicosis, lesions in organs of the immune system are consistently observed, but in this case there was no indication of immune suppression at the dosages studied (0, 3, 20 and 50 mg/cow/d). At the end of 90 d of treatment, liver selenium concentration in cows consuming 20 and 50 mg/d of selenium was 4.3 and 6.8 times greater, respectively, than cows in the unsupplemented group. With an increase in selenium intake from 50 to 100 mg/d for an additional 28 d, liver selenium content more than doubled and was 15-fold greater than in control cows at the end of the study. Serum selenium concentrations after 90 d were in all instances only 2.5 times the mean of the controls. In cattle it appears that liver is a more sensitive indicator of selenium intake above requirements than is serum.

Organic forms of selenium, such as seleno-methionine containing proteins, are apt to be more toxic than sodium selenite (Ellis et al., 1997). The chronic toxicity of seleno-methionine is lower than that of selenite (Spallholz and Rafferty, 1987). In a 30-d trial with long-tailed pregnant female macaques (Hawkes et al., 1992), the highest tolerated dose of L-seleno-methionine was estimated at 150 ug/kg. Plasma selenium associated with increased weight loss due to toxicity was > 2.8 mg/L. Both the L- and D-selenium-methionine isomers exhibited the same toxicities in rats and both isomers were retained in skeletal muscle, heart, liver and the erythrocytes to a similar degree (McAdams and Levander, 1987). It would appear preferable that L-selenium-methionine rather than the DL- isomer be employed for human selenium supplementation, although for animals the DL- mixture would be acceptable. The concern that the incorporation of selenium-methionine into body proteins could increase to toxic levels is not warranted (Patterson and Levander, 1997) because a steady state is established which prevents the uncontrolled accumulation of selenium. The release of selenium-methionine from body proteins by catabolic processes during an illness should not result in selenium toxicity because no mechanism for the selective release of selenium-methionine during catabolism exists.

However, the beneficial effects of selenium are many, and it is considered an essential nutrient. Rotruck et al. (1973) at the University of Wisconsin and Flohe et al. (1973) at Tubingen, Germany established that selenium is an integral part of erythrocyte glutathione peroxidase. The role of selenium as a defense against oxidant substances cannot be explained by selenium-glutathione peroxidase alone. In a 1980 study, selenium-deficient rats were shown to be extremely sensitive to diquat-induced liver injury (Burk et al., 1980). Doses of the compound which did not injure control rats caused massive liver necrosis and lipid peroxidation in selenium-deficient animals. A single injection of selenium given 10 h before the diquat strikingly ameliorated the injury. However, selenium injection increased glutathione peroxidase activity by only a trivial amount, indicating that selenium protected against diquat injury by a mechanism other than glutathione peroxidase.

Other closely related selenium-dependent peroxidases were soon identified in other cells and plasma. A second plasma protein secreted by the liver was named selenoprotein P. Burk and Gregory (1982) identified selenoprotein P and recognized that it accumulated in plasma. They showed that it incorporated selenium more rapidly than did glutathione peroxidase, and that it had a greater avidity for selenium than did glutathione peroxidase when the element was in short supply. Motsenbocker and Tappel (1982) have indicated that selenoprotein P is a transport protein for selenium. They cite as evidence the rapid incorporation of selenium into selenoprotein P and its appearance in the plasma before selenium accumulates in other tissues.

A selenium-dependent 5'-deiodinase was found important for the thyroid hormones in order to convert tetraiodothyronine (thyroxin) to triiodothyronine. Thus, it was established that selenium has activity as an antioxidant, in growth, and in resistance to cold stress. Also, it has been reported that selenium deficiency adversely impairs several aspects of immunity ranging from neutrophil function to resistance to infection (Boyne and Arthur, 1986; Turner and Finch, 1991; Levander et al., 1995).

Awadeh et al. (1998) have found that plasma IgM concentrations were higher ($P<0.05$) in cows given salt with 60 mg/kg selenium as Se-yeast compared to those supplemented with 60 mg/kg selenium as selenite. Calves from cows given salt with 60 ppm selenium as either selenite or Se-yeast had similar ($P >0.05$) IgM concentrations in plasma. Cows supplemented with 120 mg/kg selenium in salt mineral mix had higher ($P <0 .05$) levels of IgG in colostrum, and so their calves had higher concentrations of IgG in plasma.

Ortman et al. (1999) fed 24 Swedish Red and white dairy heifers no supplementary selenium for 6 mo. The basic diet contained 0.026 mg/kg selenium in feed dry matter. After the depletion period, the animals were divided into 4 groups; groups 1-3 received 2 mg additional selenium daily as sodium selenite, sodium selenate and a selenium yeast product, respectively. Group 4, the control, received no additional selenium. At the start of the supplementation mean concentration of selenium in plasma was 299 nmol/l, which is marginal, but after 3 mo of supplementation concentrations were adequate in the three groups receiving selenium. The concentration of selenium in plasma was 15 % higher ($P<0.05$) in the Se-yeast group than in the selenite and selenate groups but there was no difference ($P<0.05$) between selenite and selenate groups . The animals in the control group had 71 % significantly lower concentrations of selenium in plasma than those in the supplemented groups. There was no difference in the concentration of triiodothyronine ($T_3$) among any of the groups, but the concentration of thyroxine ($T_4$) was higher ($P<0.05$) in the un-supplemented control group.

Swecker et al. (1989) supplemented sodium selenite to 60 weaned calves and found that calves fed 20 mg/kg selenium in the mineral mixture ad libitum had 32 % lower antibody responses ($P<0.02$), compared with calves fed 80, 120, 160 or 200 mg/kg selenium. Calves fed the higher levels of selenium had 65 % higher ($P<0.001$) blood selenium concentrations on day 70 compared with calves fed 20 mg/kg selenium. Selenium supplementation had no effect on weight gain.

Wichtel et al. (1996b) conducted a study where two 30 g intraruminal pellets containing 10 % elemental selenium in a finely divided matrix of Fe and providing about 3 mg selenium per day were fed for at least a year. They found that supplementation with selenium increased serum selenium concentration in 20-22 mo old pregnant dairy heifers. Mean prepartum serum selenium concentration in the heifers receiving no supplementary selenium was 10 ng/ml, indicating selenium deficiency. Treatment increased prepartum serum concentrations of selenium to 74 ng/ml

(P<0.001). The postpartum serum selenium concentration remained very similar at 76 ng/ml.

In cows from 15 dairy herds, serum selenium concentrations ranged from 0.021 to 0.789 ug/ml, while 0.05 to 0.40 ug/ml is the reported range for adequate serum selenium concentrations in cattle (Stevens et al., 1985).

Bass et al. (2000) administered selenium as sodium selenite at a dose of 32.5 mg for Holstein cows and 22.5 mg for Jerseys via a single injection given on the right side of the neck. During the non-lactating period, cows had unlimited access to tall fescue, orchard grass and white clover pastures, whose selenium concentration ranged from 30 to 60 ug selenium/kg of pasture DM. At 3-4 wk prior to parturition blood concentration of selenium was 145 ng/ml for Jerseys and 108 ng/ml for Holsteins. Within 24 hours after parturition the concentrations were 155 ng/ml for Jerseys and 124 ng/ml for the Holsteins. At 2-3 wk after calving, the blood selenium concentrations were 167 ng/ml for the Jerseys and 157 ng/ml for the Holsteins, while at the end of lactation the respective concentrations were 177 and 141 ng/ml. Jerseys had significantly higher (P<0.001) selenium concentrations than Holsteins at 3-4 wk prior to calving and immediately after. This finding suggests a breed-related difference between Holsteins and Jerseys in selenium metabolism.

When Ivancic and Weiss (2001) fed mid-lactation Holstein cows diets with either 0.1 or 0.3 mg of supplemental selenium (sodium selenate) per kg of DM for 112 d, they found that selenium treatment had no effect on DMI or on milk production. Plasma selenium concentrations were similar for both treatments (63 and 64 ug/L for 0.1 and 0.3 mg/kg selenium, respectively), while milk selenium was 17 and 19 ug/L, respectively.

## Organic versus Inorganic Selenium

Inorganic sodium selenite and sodium selenate are the forms providing most of the selenium in animal feeds while manufactured and plant-derived seleno-methionine and selenium-enriched yeasts provide the organic forms (Torrent, 1997). Selenium provided in the form of yeast has at least two major advantages over inorganic compounds. First, it has higher bioavailability and second, it is never prooxidative because it is already in the organic form (Mahan, 1995). In fact, inorganic selenium may act to oxidize fats. Lipofucsin, a metabolic product of lipid peroxidation, has been found to be highest when inorganic selenium is used in the diet (Pehrson, 1993b). Other authors (Dougherty and Hoekstra, 1982; Kumpalainen et al., 1985; Levander and Burk, 1986) have also reported that inorganic selenium has prooxidative effects.

It is well known that organic selenium compounds are stored efficiently in the bodies of pigs (Sankari, 1985) and rats (Whanger and Butler, 1988), and it can be made available when the dietary supply becomes marginal (Beilstein and Whanger, 1988). In contrast, an excess of dietary inorganic selenium would be excreted largely via the urine (Fisher, 1995). Selenate is reduced to selenite which in turn is further reduced to selenide (Ganther, 1979). Selenide is methylated to form dimethyl-selenide, which is exhaled through the lungs, or trimethyl-selenomium which is excreted via the kidney (Levander, 1986 ; Janghorbani et al., 1990). There seems little to be concerned about selenium in animal diets as an environmental problem, because the unabsorbed inorganic selenium in animal feces is largely insoluble elemental selenium and metal selenides, and urinary trimethyl-selenomium is poorly available (NRC, 1983; Nriagu, 1989).

Inorganic selenium is incorporated into the selenoproteins by a selenium substitution for a sulfur atom in the cysteine residue of the protein. Organic selenium is, however, present as seleno-methionine. This seleno-methionine is nonspecifically incorporated as such into all proteins as a substitute for methionine (Gerloff, 1992), which is what probably accounts for the tendency of this form of selenium to be retained in the body (Griffiths et al., 1976). Inorganic selenium (sodium selenite) is only incorporated into a specific amino acid (cysteine) in the seleno-proteins (Burk, 1991). The stored form of organic selenium is in a non-functional state, i.e. not used immediately for the formation of biologically functional seleno-proteins (Mahan, 1994, 1995). Since this non-functional selenium is better retained than inorganic selenium, its conversion to selenide before conversion to selenocysteine and other seleno-proteins, which are functional, thus appears to be more efficient. Both ruminants and non-ruminants can absorb organic selenium easily, but the inorganic form is handled differently. In the rumen, selenite is converted to selenide which is not absorbed. The extensive rumen microbial population is, however, capable of converting a significant portion of the selenide to organic forms which are actively absorbed. In the non-ruminant, selenite is absorbed passively and the organic forms are absorbed actively (Edens, 1997). It has been reported (Koenig et al., 1991) that selenium absorption to serum and liver is 11.4 % and 10.6 %, respectively, when administered orally, and that when administered intravenously the respective absorption rates are 10.2 and 8.6 %.

After administering subcutaneous injections of sodium selenate and barium selenate to sheep, Archer and Judson (1994) reported that the response in blood and tissue selenium concentration was slow in sheep with barium selenate treatments compared to those given sodium selenate, but continued to increase for at least 112 d, indicating a sustained slow release of selenium. This can be attributed to the low solubility of barium selenate, resulting in a slow mobilization of selenium from the site of injection.

Other authors have also reported that there are differences in absorption and retention of selenium between the organic and inorganic forms (Anonymous, 1983; Levander, 1986). Ullrey et al. (1977) have reported differences in tissue selenium concentrations which were higher from diets consisting of natural sources compared to those that resulted from diets supplemented with selenite. Inorganic selenium most likely passes across the intestinal wall by passive diffusion (Anonymous, 1983; Levander, 1986; Pehrson, 1993b). Prior to intestinal absorption, a portion or most of the selenite selenium is reduced to insoluble forms by the rumen micro-organisms (Anonymous, 1983; Levander, 1986). A high-roughage diet favors much less inorganic selenium conversion into soluble forms than does a high-carbohydrate diet. In contrast, seleno-methionine likely passes across the intestinal wall by an active transport mechanism (Pehrson, 1993b). It is not known how selenocysteine and other organic selenium compounds are absorbed. However, after absorption, selenocysteine would be catabolized by selenocysteine B-lyase, thus releasing selenium (Levander and Burk, 1990). Other factors appear to interfere with the absorption of selenium from the intestinal tract. Sulfates or nitrates in water may tie up selenium and prevent absorption, and high dietary calcium may decrease intestinal absorption of selenium (Harrison and Conrad, 1984b; Smith et al., 1988).

Acevedo-Maldonado et al. (1999) assigned 70 Holstein cows at random to 5 treatments: injectable selenium and vitamin E (Mu-Se) at a rate of 1.1 ml/100 kg BW at 30 or 60 d prepartum; intraruminal bolus of slow release selenium (DuraSe-120), enough to provide 3 mg/d selenium during 120 d inserted at 60 or 120 d prepartum; and a control, without supplementary selenium. The pretreatment selenium concentrations in cow plasma were 0.078, 0.077, 0.063, 0.060 and 0.080, respectively. At parturition, the respective values were 0.114, 0.125, 0.123, and 0.090, with all treatments different ($P<0.019$) than the control but not different between themselves. At 30, 60, 90 and 120 d postpartum there were no differences detected between treatments, including the control.

In contrast to the majority of elements that are primarily absorbed from the gastro-intestinal tract in an inorganic ion form, selenium may be absorbed in an inorganic form such as selenite or in an organic form as a component of seleno-amino acids (Koenig et al., 1997). Bioavailability of seleno-methionine and Se-yeast was determined by Smith and Picciano (1987), relative to that of an inorganic form, selenite, in the lactating rat. After being on a selenium-deficient diet during pregnancy, groups of rats were fed experimental diets containing either 0.1, 0.25 or 0.5 mg/kg selenium as selenite, seleno-methionine or Se-yeast. Dams fed seleno-methionine had 18 % higher ($P<0.05$) plasma selenium concentrations than dams fed selenite. Liver selenium concentrations were 24 % higher ($P<0.05$) in dams fed seleno-methionine than in both other groups. Pups of dams fed seleno-methionine or Se-yeast had plasma selenium concentrations 27 % higher ($P<0.05$) than selenite pups. Liver selenium concentrations were 31 % higher ($P<0.05$) for pups of dams fed seleno-methionine than for those of dams fed Se-yeast, which in turn were 42 % higher ($P<0.05$) than for those of dams fed selenite. In this study the bioavailability of Se-yeast fell between that of selenomethionine and selenite. The greater bioavailability of seleno-methionine and Se-yeast to pup tissues may be a direct result of the greater selenium concentrations in the milk of their dams.

Conrad and Moxon (1979) compared sodium selenite and brewers grain as sources of selenium. The dietary selenium treatments were as follows: 1) control diet (0.78 mg/d total Se); 2) 0.1 mg/kg selenium from selenite (2.53 mg/d total Se);

3) 0.2 mg/kg selenium from selenite (5.08 mg/d total Se; 4) selenium from brewers grain (2.86 mg/d total Se) and, 5) selenium from brewers grain plus 0.5 mg/kg added selenium from selenite. At days 0, 42 and 91 of selenium supplementation, plasma selenium concentrations were 32, 31 and 29; 31, 67 and 67; 30, 77 and 70; 27, 96 and 94 and 24, 107 and 109 ng/ml, respectively, for treatments 1, 2, 3, 4 and 5. The concentrations of selenium in milk for days 0, 42 and 91 of selenium supplementation were 10, 8 and 10; 11, 12 and 17; 7, 14 and 17; 8, 33 and 29 and of 8, 42 and 37 ng/ml , respectively, for treatments 1, 2, 3, 4 and 5.

Harrison and Conrad (1984a) evaluated selenium absorption and retention in mature non-lactating Holstein cows when sources of selenium were linseed meal and sodium selenate. They found that selenium from linseed meal was absorbed at a rate of 37 % and 74 % of this selenium was retained. For sodium selenate, absorption rate was 48 % and 77 % of the selenium was retained. It was concluded that selenium absorption and retention are linear when dietary selenium intake ranges from 400 to 3100 ug/day. Zero retention of selenium could occur with selenium intake less than 332 ug/day. The practical significance of these results is that cows consuming <10 kg dry matter/day, with selenium content of < 0.3 ug/g dry matter, could be in negative selenium balance.

Nicholson et al. (1991a) supplemented growing beef and dairy cattle with inorganic selenium (sodium selenite to supply 1 mg Se $kg^{-1}$ of supplement) and organic selenium (Alkosel yeast to supply 1 mg Se $kg^{-1}$ of supplement) plus a control (no supplemental selenium) with the following results: there were no differences ($P>0.05$) in weight gains or efficiency of feed conversion due to treatment, and blood selenium concentration was higher ($P<0.01$) for the organic (141 ug/L) than for the inorganic (102 ug/L) treatment and the control (45 ug/L). These results show there are no differences among dairy and beef cattle in response to selenium supplementation, that selenium supplementation had no effect on animal performance, that more than 4 mo are required to fully deplete or replete blood selenium and that supplemental organic selenium gives higher ($P<0.01$) blood selenium concentrations than does inorganic selenium.

In another study, Nicholson et al. (1991b) sprayed alfalfa with an aqueous solution of sodium selenate (20 g Se in 200 L of water $ha^{-1}$) and supplemented selenium in the feed. Silage was made from the alfalfa (802 $ug/kg^{-1}$ of selenium, DM) and the concentrate feed contained 398 ug/kg selenium. There were no significant effects due to treatment on milk production. Milk selenium concentrations were (ug/L) 30 for the control (no added selenium), 35 for the feed and 58 for the alfalfa silage. The cows receiving the selenium fertilized alfalfa silage averaged higher ($P<0.01$) milk selenium than those on the other two treatments. These same cows also had higher ($P<0.01$) plasma concentrations (111 ug/L) than the other groups (88 ug/L for the feed and 75 ug/L for controls).

## Selenium and Thyroid Hormone Metabolism

The effects of selenium deficiency on thyroid hormone metabolism occur before growth or food intake are affected, suggesting that they occur by different mechanisms than those that affect the hormones in malnutrition and illness (Arthur, 1991). The effects of a selenium deficiency are unlikely to be secondary consequences of changes in antioxidant status since vitamin E deficiency does not change plasma $T_4$ and $T_3$ concentrations (Beckett et al., 1989, 1990).

The impaired performance of ruminants grazing forages deficient in selenium may in part be the result of alterations in thyroid hormone metabolism induced by selenium deficiency (Andrews et al., 1968). The activity of the seleno-enzyme type I triiodothyronine 5'-deiodinase is sensitive to changes in selenium intake (Beckett et al., 1993). Selenium supplementation causes plasma concentrations of triiodothyronine to be increased and thyroxine to be decreased in ewes (Donald et al., 1993) and calves (Wichtel et al., 1996a) grazing pastures deficient in selenium.

In selenium deficient rats and cattle, plasma concentrations of the hormone $T_4$ increase and those of 3,5,3'-triiodothyronine ($T_3$), decrease (Beckett et al., 1987, 1989; Arthur et al.,1988, 1990). Plasma free $T_4$ concentrations can increase three-to-four-fold in selenium deficiency (Arthur et al., 1990). Thus, it seems that changes in plasma thyroid hormones are a specific response to selenium deficiency. Up to 80 % of the $T_3$ in the plasma is formed by mono-deiodination of $T_4$ in nonthyroidal tissues (Leonard and Visser, 1986). This reaction is induced by two iso-enzymes of iodothyronine 5'- deiodinase which are different in their properties, tissue distribution, sensitivity to inhibitors and specificity for substrate (Safran and Leonard, 1991).

Type I deiodinase in liver and kidney is the major source of plasma $T_3$, whereas type II deiodinase, which is found in the central nervous system, pituitary, and brown adipose tissue, contributes little to circulating $T_3$ but is the major source of intracellular hormone in these tissues (Leonard and Visser, 1986). In a selenium deficiency, the increase in plasma $T_4$ and decrease in $T_3$ plasma concentrations are consistent with the impairment of tissue deiodinase activities.

That the effects of selenium deficiency on thyroid hormone metabolism are mediated through the iodothyronine deiodinases and that these enzymes are seleno-proteins or dependent on selenium containing co-factors for their activity has been postulated by Beckett et al. (1987, 1989), Arthur et al. (1987), and by Arthur and Beckett (1989). The impaired thyroid hormone metabolism may be responsible for decreased growth and resistance to cold stress in selenium-deficient animals. The daily liveweight gain of Angora goats that received supplementary selenium (intra-ruminal pellets) was 26 % higher than that of goats that received no selenium ($P<0.001$) during the final month of a study (Wichtel et al., 1996c). When measured over the entire 95-day study period, however, the live-weight gain was not significantly higher for goats that received supplementary selenium.

Arthur et al. (1988) demonstrated that consumption of a selenium deficient diet by Friesian steers during 23 weeks had a decreasing effect on whole blood selenium concentrations to 0.008 mg/L compared to 0.081 mg/L in the selenium sufficient group. However, during the 23 weeks the weight gains were similar at 162.4 kg (selenium deficient group) and 161.6 kg (selenium-sufficient group). Also, after 20 and 23 weeks plasma $T_4$ concentrations were higher in selenium-deficient animals (62 vs 50 %, respectively). In contrast, plasma $T_3$ was decreased less at 30 vs 35 %, respectively, for selenium adequate and deficient animals. The selenium sufficient group of calves were being supplemented with 0.1 mg $kg^{-1}$ of diet as sodium selenite. The $T_3$ concentrations in calves is important because $T_3$ enhances the synthesis of uncoupling protein, necessary for brown adipose tissue thermogenesis (Carstens, 1994).

Wichtel et al. (1996a) report that selenium supplementation increased plasma $T_3$ while it decreased plasma $T_4$ concentrations of Holstein calves in two herds. The supplement consisted of two intra-ruminal pellets which released selenium at a rate of 3 mg/day for at least one year. This has also been shown in other species. A selenium deficiency in chicks decreases circulating $T_3$ concentrations whereas selenium supplementation increased the concentration in plasma (Jensen et al., 1986). Selenium deficiency in rats decreased the conversion of $T_4$ to $T_3$ in liver homogenates (Beckett et al., 1987; Arthur et al., 1988; Arthur, 1991) In selenium deficient rats plasma $T_4$ concentrations were increased and those of $T_3$ were decreased. Guo et al. (1998) concluded that thyroid hormone metabolism can be regulated by selenium through iodothyronine deiodinase, after studying the nutritional inter-relationships between selenium and iodine in broiler chicks.

Because of the large quantitative differences in concentrations of $T_3$ and $T_4$ in blood , significant decreases can occur in $T_3$ without detectable changes in $T_4$. Thus, concentrations of $T_3$ in plasma of cows given salt with only 20 mg/kg selenium as selenite averaged 86 % of those of cows supplemented with 60 mg/kg selenium as selenite or selenium-yeast (Awadeh et al., 1998). The concentrations of $T_3$ in plasma of selenium-deficient guinea pigs were 69 % of those in guinea pigs fed selenium-adequate diets (Cammack et al., 1995). Similarly, selenium-deficient rats had $T_3$ concentrations in plasma that were reduced 23 % (Thompson et al., 1995). Calves born to cows supplemented with 60 mg/kg selenium as selenium-yeast had higher plasma $T_3$ concentrations at birth than calves supplemented with 20 and 60 mg/kg selenium as selenite (Awadeh et al., 1998). In Angora goats, supplementation with selenium (intra-ruminal pellets) decreased ($P<0.001$) plasma total thyroxine concentration by 18 %, but the small increase in plasma total triiodothyronine was not significant, as reported by Wichtel et al. (1996c), who concluded that the dietary selenium requirement for growth of the goats is greater than 0.02 mg/ kg DM, and that a serum selenium concentration of less than 140 nmol/litre reflects an inadequate status. A minimum serum selenium concentration of 150 nmol/litre is recommended for calves grazing pasture (Ellison and Feyter, 1988).

If production responses to selenium are mediated via the thyroid axis, changes in the tissue concentration of triiodothyronine, the most active of the thyroid hormones, would be expected (Wichtel et al., 1996c). It would appear, however, that the circulating triiodothyronine is not as sensitive as thyroxine to a deficiency of selenium, possibly because of changes in the secretion and catabolism of triiodothyronine that occur in response to selenium deficiency (Chanoine et al., 1992).

## Selenium Supplementation of Grazing Animals

Most livestock regions in the USA, including the Southeastern states, are deficient in selenium and result in selenium responsive diseases when cattle are not supplemented (Kubota et al., 1967). However, decreased productivity resulting from marginal selenium deficiency may be present in larger areas. A marginal deficiency may be underestimated because clinical signs do not present themselves and the deficiency may not present itself every year and in all regions (Wilkins and Kilgour, 1982; Langlands et al., 1991).

Selenium is not required by plants but is an essential trace element for adequate nutrition and health of fish, birds, animals and humans (Mayland, 1994). Plants, while not requiring selenium, absorb it from the soil solution and cycle it to ingesting animals (Mayland, 1994). In general, diets containing 0.1-0.3 mg/kg selenium will supply adequate selenium for all these species. Many soils are incapable of providing that amount to plants growing on them. Effects on cattle health have been associated with deficiency and may include, according to Maas (1983) and Corah (1991): myodegeneration, retained placenta, infertility, abortions, birth of premature, weak or dead calves, cystic ovaries, metritis, delayed conception, erratic, weak or silent heat periods, poor fertilization rates, diarrhea, decreased immune response, ill “thrift” and death.

The chemical form of inorganic selenium in the soil depends largely on the pH and the oxidation-reduction potential of the soil (Ylaranta, 1993) Three oxidation states of selenium are stable within the range of oxidation-reduction potential of the soil existing in the environment: +6 (selenate), +4 (selenite) and 0 (elemental selenium). The predominant mobile inorganic forms of selenium are selenate in aerated alkaline soils, and selenite in acid, reducing soils such as those that generally occur in humid areas (Geering et al., 1968). Clay minerals, calcite, Al and Fe oxides are the selenium-sorbing mineral constituents in the soil (Goldberg and Glaubig, 1988). Foliar spraying with selenate is also suitable for use in practice, in addition to selenate fertilizer. The effect of spraying with selenite on the selenium content of crops depends largely on the stage of growth of plants at time of spraying. Selenate is more suitable for this because it can be sprayed on anytime, and it is not always possible to choose the spraying time exactly and consistently. Both sources of selenium are equally effective in raising the selenium content in grasses like timothy (Ylaranta, 1993). In acid soils, selenite fixes quite rapidly into a form that is poorly available to plants. Sorption of selenite by soil seems to be weaker, however, than that of phosphorus (Ylaranta, 1983b). Selenium added to the soil in the form of selenate remains in a readily soluble form for several months. On coarse mineral soils and soils with a high humus content treatment with selenate had a marked residual effect on the selenium content of barley, indicating that some of the selenium from the selenate remains available to the plants for at least two growing seasons (Ylaranta,1985). From field experiments, the selenate uptake of grasses is about 20 %. The selenium fertilization method practiced in Finland indicates that apparently the selenium uptake of plants is clearly higher than experimental field results (Ylaranta, 1990). In general, soils with less than 0.6 mg/kg selenium are likely to produce crops with insufficient selenium (<0.1 mg/kg) to protect livestock against selenium deficiency (Gupta and Gupta, 2000).

Ekholm et al. (1991) fed 32 bulls from birth to 13-14 mo with rations containing either 0.03 mg selenium/kg (basic level in local feeds), 0.25 mg inorganic selenium as sodium selenite, or 0.25 or 0.40 mg/kg plant selenium in dry matter from forage produced by spraying with sodium selenite. The maximum response was caused by the diet containing 0.40 mg/kg plant selenium (1.7 mg/kg vs. 0.23 mg/kg for the control). Plant selenium at both concentration levels was more effective than sodium selenite in raising the selenium content of muscle tissues. The selenium concentration of liver increased more than that of other organs in all dietary groups. Muscle tissue and liver were high responders to plant selenium (increased 7.4-12.0 fold in highest plant selenium treatment). Prasad and Arora (1991) have also reported that organic selenium sources seem to accumulate more in tissues than inorganic sources.

The most important methods of supplementing selenium to grazing animals include: 1) a free-choice selenium mineral mix, 2) selenium injections (sub-cutaneous and intra-muscular), 3) selenium drenches, 4) selenium ruminal pellets, and 5) selenium fertilization.

In Norway, up until 1980, selenium was usually administered to cattle by injection or by oral administration to ruminants of high, single doses of sodium selenite or sodium hydro-selenite. The toxicity risk of this regimen is relatively high, which was proved in practice by several incidences of often fatal selenium intoxication. Then from 1980, by official directives of 1979, selenium has been added to compound concentrates and mineral mixtures to prevent deficiency diseases. This has reduced the incidence of selenium deficiency diseases to a minimum, and also has had an impact on animal products (Overnes, 1993). When selenium is mixed into feed the selenium requirements in lambs will be close to 0.3 mg selenium/kg feed (Moksnes and Norheim, 1983). When given through mineral mixtures or salt licks, the ewes should receive approximately 400 ug selenium/day. This will result in sufficient selenium levels in blood, milk and offspring to protect against selenium deficiency diseases. This seems to be achieved with mineral mixtures and salt licks containing 20-25 mg selenium/kg (Overnes et al., 1985b). The injection of a long-acting barium selenate preparation may be a good alternative. The dosage used once, approximately 1.2 mg selenium/kg body weight, is sufficient to maintain an adequate selenium status for as long as two consecutive lambing seasons (Overnes et al., 1985a). An alternative to a depot injection is the use of ruminal pellets, either in the form of iron bullets with a grinder or as glass-boli, the latter often used in a combination of selenium, copper and cobalt (Judson et al., 1991). The safety and efficacy of these methods seem far superior to single doses of sodium selenite. Another way of increasing the selenium supply in ruminants is by using selenium-enriched NPK fertilizer, which will result in increased forage selenium concentration (Overnes et al., 1986).

McDowell (1992) indicates that selenium mineral mixes fed free-choice present some disadvantage in that a few animals consume little or nothing at all, even though this is the most utilized method. According to Miller and Andrews (1964), drenches are only effective for up to 6 weeks and require considerable labor, and intraruminal pellets are effective for a longer period of time, but this is highly variable and they are commonly regurgitated. Direct administration of selenium by injection gives satisfactory results, especially with slow-release long lasting products (Langlands et al., 1990; Van Ryssen et al., 1992). However, these methods are expensive and require handling of the animals.

The provision of selenium through a fertilizer would be uniformly received by the animals consuming the forage, and the topdressing of pastures with selenium, usually mixed with other nutrients, has proven to be effective. This practice is common in the high rainfall areas of Australia (Whelan, 1994) as it is in livestock production in New Zealand (Watkinson, 1983), and in Finland it is the almost exclusive way of selenium supplementation (Gissel-Nielsen, 1993). Selenium-supplemented fertilizers were first used in Finland in the 1985 growing season. A level of 6 mg/kg selenium (as selenate) is used in fertilizers for forage production. The level of selenium in forages (mg/kg DM) varies according to the type of forage. Thus, from 1986 to 1991 it has fluctuated from 0.12 to 0.19 in silage, from 0.07 to 0.15 in hay, from 0.11 to 0.23 in barley, from 0.12 to 0.26 in oats, and between 0.22 to 0.30 in commercial feed.

It has been reported (Grant and Shepard, 1983) that different grass species do not seem to vary widely in their selenium content in the same type of soil. However, Van Ryssen et al. (1998) found that ryegrass seems to have a much better ability than kikuyo to take up selenium from the soil. Several authors have reported similar differences between other grass species (Davies and Watkinson, 1966; Grace and Clark, 1991; Mayland, 1994). Plants vary in their uptake of selenium, but this nutrient is not a requirement for plant growth. Slower growing and deeply rooted species usually contain slightly higher concentrations of selenium. On moderately low selenium soils, alfalfa accumulates more selenium than many other forage plants (Mayland et al., 1989). This may be related to differences in rooting depth and to genetic traits that may affect absorption and translocation of selenium to shoots. Gupta and Watkinson (1985) indicated that improvement of soils and pastures can affect trace element availability to the plant and animal in several ways. Increased soil fertility and aeration can decrease the concentrations of selenium through the mineral-diluting effect of rapid pasture growth. Higgins and Fey (1993) add that uptake of selenium is affected by soil properties such as pH, clay content and also the concentration of competing ions, such as $S0_4{}^{2-}$, with the selenium being more available to the plant at a higher pH.

McQuinn et al. (1991) indicate that selenium concentrations could be increased by 19 % in tall fescue (*Festuca arundinacea*) through genetic selection. This species is adapted to most of the selenium deficient pastoral areas in the United States. Genetic selection in this forage species promises to increase herbage selenium levels and satisfy the selenium needs of grazing animals in marginally deficient areas. It is possible that similar breeding opportunities may exist in other grasses.

The selenate form of selenium, as compared to selenite, is best for use in fertilization and both sodium and barium selenate have also been used. Only a tenth as much selenate is required, in contrast to selenite, to produce a given result (Oldfield, 1998). Sodium selenate is much more available to plants than the more reduced selenite form (Ylaranta, 1983a). According to Cary and Allaway (1973), however, a 2 kg/ha application of sodium selenite to slightly-acid, medium-textured, low-selenium soils would ensure levels of selenium in forage that would protect livestock against selenium-deficiency diseases for at least 4 years. Also, the safety of selenite is much greater than that of selenate, and these authors suggest that comparable levels of selenate would produce plants containing toxic levels of selenium.

It is known that organic forms of selenium are more bioavailable than the inorganic forms (Pehrson et al. 1989). Most of the selenium produced naturally in plants is in organic combination, and this suggests that the use of inorganic selenium as a fertilizer amendment will be beneficial through raising the contents of organic selenium in feeds and foods (Mahan, 1999; Oldfield, 1999).

In Finland, after starting a program of forage fertilization with selenium (6 mg/kg fertilizer from sodium selenate), Varo (1993) reported milk selenium concentrations rose from 30 ug/kg DM to a plateau of 175 ug/kg. The concentration of selenium in cheese (430 ug/kg) almost doubled that in milk due to its higher protein content. Initially, the selenium content of grass was around 10 ug/kg DM, but after the fertilization program started this concentration rose to 150 ug/kg.

Plants retain selenium in protein as seleno-aminoacids and therefore plants and their parts higher in protein tend to be higher in selenium. Among crops, brassicas and legumes, particularly soybeans, contain higher selenium than other crops (Gupta and Gupta, 2000).

## Use of Selenium in Cow-Calf Herd Supplementation

Awadeh et al.(1998) report that supplementation of cows with selenium did not affect calf birth weights in year 1 or weight gains of calves in either year 1 or 2, after conducting a two year study. Also, selenium supplementation of dams did not increase calf birth weights as reported by Ammerman et al., (1980) and Hidiroglu (1987). More recently, Wichtel et al.(1996a) conducted two trials on selenium supplementation of grazing calves (5 mo of age) and found a positive weight gain response in one herd but not in the other. Other authors, Ullrey et al. (1977), Swecker et al. (1989) and Lacetera et al.. (1996) all have found no significant impact of selenium on body weight gain. However, Donald et al. (1994) reported that at the end of a six month study, selenium supplemented sheep (intraruminal pellet) were heavier ($P<0.05$) than un-supplemented sheep (36.1 vs. 32.4 kg). Selenium supplementation increased ($P<0.01$) daily liveweight gain from 42 to 61 g/day. In another study, Donald et al. (1994) supplemented ewes grazing selenium-deficient pastures with 0-8 mg selenium as sodium selenate every 14 d and found that selenium supplementation of the dam increased plasma selenium concentrations in the ewe and in the lamb. Birth weight was not affected by selenium treatment. Masters and Peter (1990) reported that the use of selenium pellets and glass boluses resulted in increases ($P<0.01$) in liver and plasma selenium of sheep after 26 wk, but the effect of the treatment did not persist to 54 wk in sheep given selenium pellets, whereas sheep given glass boluses still had higher ($P<0.01$) selenium in both plasma and liver. Pehrson (1993a) indicated that the growth rate of young ruminants may be reduced at severe levels of selenium deficiency. At marginal or optimal dietary selenium levels it can be assumed that there is no significant reduction.

Serum selenium concentrations of 70 to 100 ng/ml are a fairly reliable indicator of selenium adequacy and are probably necessary to be confident of avoiding subclinical selenium deficiency (Gerloff, 1992). Smith et al. (1988) also recommend a concentration of 0.07 ug/ml in plasma or serum of cattle as an indicator of selenium sufficiency. Dietary intakes of 0.3 mg of supplemental selenium/kg of DM, or 6 mg of selenium per cow per day seem to be necessary to achieve the desired serum selenium concentration in most cases. There is great variability in herd response to supplementation and this may be due to several dietary and management factors, such as: 1) dietary concentration of interfering elements such as Ca, S, or trace minerals, 2) dietary and management effects on the reducing environment of the rumen such as amount of concentrate or feeding pattern of the diet, and 3) selenium concentration of the basal feedstuffs, affected by variation in soil characteristics and selenium content, as well as differences in soil and plant management among farms.

Toledo and Perry (1985) conducted an experiment to evaluate amounts and methods of selenium administration, namely, oral supplementation (1 vs 2 mg/head/d as sodium selenite, last 60 d of gestation) and intramuscular injection (50 mg selenium at 40 and 29 d prepartum vs 50 mg selenium at 60, 40 and 20 d prepartum) plus a control (no selenium). Initially, (60 d pre-partum) selenium in blood serum averaged 0.023 mg/L. Control cows did not change significantly through 20 d postpartum. Cows fed 1 mg selenium/d over the same period showed no difference in serum selenium except at 20 (0.032 mg/L) and 10 d (0.034 mg/L) prepartum. However, cows fed 2 mg selenium/d had selenium in serum higher ($P<0.05$) than at the start of the experiment and also higher than control cows ($P<0.05$) 30 d prepartum (0.042 mg/L) and through parturition (0.037 mg/L). Cows administered two intramuscular injections 40 and 20 d prepartum showed an increase ($P<0.01$) in serum selenium within 10 d (0.049 mg/L) of each injection. Cows that received three injections, at 60, 40 and 20 d prepartum, showed a response in serum selenium similar

(0.052 mg/L) to those given only two. However, those administered three injections showed a higher ($P<0.05$) response and for a longer period, since the first dosage was administered 20 d sooner than for those that received two injections. Ten days postpartum the concentration of serum selenium for the cows given three injections had decreased ($P<0.05$) and was lower than at parturition, but still higher ($P<0.05$) than at the time of initiation of the treatments. Selenium concentrations in colostrum were the same for the control and oral treatments (0.0056 mg/L) while the injected treatments had similar concentrations (0.0063 mg/L). Calves from cows that received selenium by intramuscular injection had higher ($P<0.05$) serum selenium at birth (0.037 mg/L) than those from the control cows (0.029 mg/L). The calves from cows with oral selenium supplementation had similar serum selenium concentrations than to calves whose dams received no selenium (0.029 vs 0.025 mg/L).

McDowell et al. (1990) conducted an experiment for 22 months to determine the selenium status of grazing Brahman cows and their calves as affected by supplementation with selenium and season of the year. Three treatments were administered to grazing cows for 120 days (November to March) either orally in a concentrate mixture or yearly intramuscular injection, and these were: 1) control, no selenium 2) 0.25 mg/kg selenium in mixture + intramuscular injection of 5 mg selenium + 1,500 mg vit E, and 3) 0.25 mg/kg selenium in mixture. Mean liver selenium concentrations were 0.25, 0.63 and 0.54 mg/kg (DM basis) for control, injectable and dietary selenium and dietary selenium, respectively. Treatments were higher ($P<0.05$) than the control. For serum, the mean selenium concentrations were 0.024, 0.055 and 0.045, respectively, with treatments also higher ($P<0.05$) than the control. Colostrum selenium concentrations were 0.015, 0.049 and 0.031 ug/ml and differed ($P<0.05$) among treatments. The milk selenium concentration of injectable and dietary selenium treatment was higher ($P<0.05$) than the control and dietary selenium treatment, which did not differ among themselves, while calf serum selenium concentrations were the same among treatments but higher ($P<0.05$) than the control.

Swecker et al. (1995) reported that selenium supplementation with sodium selenite to cows provided 120 mg of selenium/kg of salt mineral mix ad libitum effectively, practically and safely increased selenium status of selenium-deficient cows and their calves that grazed a selenium-deficient pasture.

In ruminants, a reduction in plasma selenium concentrations has been reported at high stocking rates (Langlands et al., 1991). This is consistent with previous observations (Langlands et al., 1982) and may have several causes. These include greater losses of selenium from the soil-plant-animal system when more plant selenium passes through the animal, as it does at higher stocking rates because of either increased selenium volatilization or a change in its chemical form to one less available to plants or more readily leached. Also, selenium uptake by plants may be reduced by botanical changes or a lower root volume in heavily grazed pastures. Presumably, selenium is more readily leached from the soil when rainfall is high, pasture growth may be increased, and selenium in the pasture on offer and in the diet diluted by the additional growth.

Langlands et al. (1990) derived relationships between selenium concentrations in blood and plasma of lambs and their dams, and calculated that the predicted blood selenium concentration in the lamb at birth was 71 % of that in the dam. In sheep supplemented with either 3, 6, or 9 mg/kg selenium (sodium selenite) on an as-fed basis for 10, 20 or 30 d, Echevarria et al. (1988) found that liver selenium concentration increased ($P<0.01$) from 0.85 to an average of 8.30 mg/kg selenium as supplemental selenium increased, and liver selenium from sheep fed control diets was lower ($P<0.01$) than that of sheep fed added selenium. Sheep fed for 30 d accumulated more ($P<0.01$) selenium in liver than those fed for 10 or 20 d. Serum selenium increased ($P<0.01$) from 0.09 ug/ml in the un-supplemented controls to 0.25 ug/ml for sheep fed 9 mg/kg added selenium. In another study, Henry et al. (1988) reported that after feeding for 10 d, liver selenium increased from 1.30 mg/kg in un-supplemented controls to 10.46, 10.08 and 9.95 mg/kg selenium (dry basis) from sodium selenite, calcium selenite or sodium selenate, respectively, when sheep were fed 9 mg/kg added selenium for 10 d . Serum selenium also increased, from 0.09 ug/ml in un-supplemented controls to 0.35, 0.22 and 0.32 ug/ml from sodium selenite, calcium selenite or sodium selenate, respectively, when fed the same amount of added selenium (9 mg/kg).

Measurements of blood selenium concentrations and live weights of pure and crossbred Merino lambs born to ewes which were supplemented or not supplemented with intra-ruminal selenium pellets and grazed at 6.3 or 12.5 ewes/ha for 4 consecutive years were taken by Langlands et al. (1990). They found that live weights at birth ($P<0.01$), in mid-lactation ($P<0.001$), and at weaning ($P<0.001$) were significantly increased in lambs born to selenium-supplemented and crossbred ewes, and in lambs born as singletons. The response in liveweight gains following selenium supplementation was greater in lambs reared at the higher than at the lower stocking rate. Lambs born to supplemented ewes initially had high selenium status as a result of placental transfer, but this status declined as lambs became increasingly dependent on pasture. Plasma selenium concentrations of lambs born to supplemented ewes were greater at weaning (0.012 ug/ml selenium) than corresponding values for the unsupplemented group (0.004 ug/ml). Number of lambs surviving per ewe mated (Langlands et al. 1991) was increased ($P<0.05$) by selenium supplementation at the high stocking rate (0.74 vs 0.86 lambs) but not at the low stocking rate (0.84 vs 0.83). The selenium x stocking rate interaction approached significance ($P<0.09$) and this suggests that selenium supplementation increased overall reproductive performance at high (0.81 vs 1.04) but not at low (1.08 vs 1.00) stocking rates.

Plasma selenium concentrations of Holstein cows were similar and averaged 0.081, 0.112 and 0.119 ug/ml at weeks 1, 7 and 13, respectively, where the animals were fed diets containing 0.2, 0.3, 0.4 and 0.7 mg/kg selenium as sodium selenite. (Maus et al., 1980).

Awadeh et al. (1998) reported that when cows were grazing selenium deficient forages (0.07 mg/kg selenium dry matter), Se-yeast was more effective than selenite in raising blood selenium concentrations after 22 months of supplementation. Blood selenium was highest at birth for calves of cows given salt with 60 mg/kg selenium as Se-yeast , but by two months of age only calves who consumed salt with 120 mg/kg selenium as selenite had elevated blood selenium. However, Nicholson et al. (1991a) reported that organic selenium was more effective than inorganic in raising blood selenium concentrations in calves at 4 months of age and similar results were reported by Chavez (1989).

Awadeh et al. (1997) reported that cows given salt with 20, 60 and 120 mg/kg selenium as selenite or 60 ppm selenium as Se-yeast consumed 1.27, 3.98, 8.57 and 4.15 mg selenium per day, respectively. The blood concentration of selenium declined in blood of cows post-partum, except for cows given salt with 120 ug/g. This post-partum reduction could be due to loss of selenium in milk during the lactation period or to repletion of tissue selenium from the fetal drain during gestation. Only the largest selenium intake was sufficient to maintain concentrations of selenium in blood during the 9 month postpartum period. Thus, cows with access to salt with 60 mg/kg selenium or less, may have transferred body selenium reserves to milk. Previously, Abdelrahman and Kincaid (1995) reported cows with daily intakes of approximately

1 mg selenium/day were unable to maintain selenium concentrations in blood during late gestation.

Some of the differences encountered between studies may be due to age of the animals, selenium intakes, and duration of supplementation (Awadeh et al., 1998). Seleno-methionine is thought to be randomly substituted for methionine in protein and to accumulate mainly in liver and muscle, according to Chavez (1989).

The selenium concentration of milk is linearly dependant on and readily indicates changes in dietary selenium. Maximum values are reached in about 40 days after the start of supplementation whereas the decrease is somewhat slower (Conrad and Moxon, 1979; Aspila, 1991). During the first years of selenium fertilization in Finland (Ekholm et al., 1993) the selenium content in milk fluctuated with the season, being about 15 % lower in the outdoor season than in winter. The seasonal variation became smaller in later years. By 1989 the fluctuation was very slight and in 1990 it was almost undetectable. The concentration in milk varied from 0.15 to 0.25mg/L selenium (DM basis) .

The selenium content of milk from a group of dairy cows increased from 0.011 to a high but constant level of 0.10-0.11 mg/L in three weeks after start of feeding a diet with selenium yeast (0.4 mg/kg in 20 kg DM), as reported by Suoranta et al. (1993). From week 8, the addition of 0.1 mg/kg selenium from selenium yeast to a diet of 16 g inactive dried brewers yeast with flour was reflected in an increase from 0.008 to a sufficient level of 0.025 mg/l in milk of another group of cows.

Selenium content of cow milk was found by Debski et al. (1987) to be lower than that of either human or goat milk. Regardless of source, less than 3 % of total milk selenium was associated with the lipid fraction. Selenium within the 120,000 x g supernatant accounted for 72, 62 and 30 % of the total in cow, human and goat milk, respectively. The quantity of selenium in milk is influenced by the dietary intakes of this trace element (Hadjimarkos and Bonhorst, 1961; Bisbjerg et al., 1970; Maus et al., 1980). A linear correlation was observed between selenium concentration in feed and content of the trace element in milk (Bisbjerg et al., 1970). Concentration alone does not ensure bioavailability of selenium. Alexander et al. (1983) reported that although tuna had a higher concentration of selenium than wheat flour, the ability of wheat flour to replenish muscle selenium was twice that of tuna.

The concentration of selenium in cow's milk varies greatly with the selenium intake of the animal (Levander, 1986). Allaway et al. (1968 ) have reported that cow's milk from a low-selenium area in Oregon contained <20 ng/ml, compared with 50 ng/ml from a high selenium area in South Dakota. Higher levels, ranging between 160 and 1270 ng/ml, have been observed for cows milk from high-selenium areas in the United States. The selenium level in milk is readily raised by supplementation of animal diets. Grant and Wilson (1968) obtained substantial milk selenium increases over a period of 3 to 4 weeks from cows receiving a single oral dose or subcutaneous dose of 50 mg selenium as selenate, while levels in untreated cows remained steady at 3 to 4 ng/ml. Selenium concentration of milk of ewes fed a low selenium diet was tripled by supplementing the diet with 2.25 mg per day selenium as sodium selenite (Gardner and Hogue, 1967).

The selenium level in feed, 0.15 to 0.20 mg selenium/kg DM, seemed to be sufficient for milk production, as indicated by Syrjala-Qvist and Aspila (1993). The highest levels of supplementation used in this study (1.81, 0.78 and 0.68 mg/kg) tended to decrease feed intake and, consequently, milk production. Selenium given in fertilized silage raised the level in milk to a greater extent than the same amount of selenium given in inorganic form. In cows receiving selenium in silage the proportion of selenium secreted into milk averaged 8 %, whereas the corresponding values for the animals given sodium selenite was 5%. With both sources, the secretion percentage diminished as selenium concentration in feed increased. In a previous study, Conrad and Moxon (1979) found that when cows on a silage-grain diet were given a selenium supplement as sodium selenite, less than 5 % of the added selenium was transferred to milk. When selenium was given in organic form bound to the feed , 19 % of selenium supplement was transferred to milk.

Pehrson and Johnsson (1985 ) have shown that two oral administrations of 30 mg of selenium as selenite during late pregnancy maintained a normal selenium status in calves for 2 to 3 months. More recently, Enjalbert et al. (1999) provided prepartum supplementation of cows consisting of 32.5 or 45.5 mg/d of selenium as sodium selenite compared with 13.0 mg/d. At 17 and 88 days of age the selenium status of calves was still satisfactory for the 32.5 and 45.5 mg selenium/d treatments. When serum selenium concentrations are deficient, the increase of selenium concentration in milk after selenium supplementation is of a very low magnitude 7 days postpartum (Stowe et al. 1988). Milk selenium concentrations have been shown to increase linearly with selenium intake of 2 and 6 mg/d in cows that had normal plasma selenium values (Maus et al. 1980). These authors reported average milk selenium concentrations of 0.034, 0.050 and 0.054 ug/ml at 1, 7 and 13 wk of supplementing Holstein cows with diets containing 0.2, 0.3, 0.4 and 0.7 mg/kg selenium. There were no differences among treatments. Norton and McCarthy (1986) have reported a 28 % increase in milk selenium concentration after selenium injection of ewes 18 days postpartum. Selenium transport into milk and colostrum occurs primarily through the secretory vesicle associated with casein in mammary alveolar epithelial cells (Allen and Miller, 1981).

Milk from cows receiving selenium supplementation has varying selenium concentrations. In beef cows (Ammerman et al. 1980), observed values decreasing from approximately 0.015 mg/L two weeks after calving to 0.010 mg/L at eight weeks after calving, with no differences among them. In dairy cows, Maus et al. (1980) reported values of 0.029 and 0.064 mg/L in mid-lactation. Similar values (0.04 to 0.06 mg/L) have been found in colostrum (Abdelrahman and Kincaid, 1995). Selenium concentrations were highest ($P<0.01$) in colostrum of cows given salt with 120 mg/kg selenium as selenite versus those given 60 mg/kg as selenomethionine from Se-yeast (Awadeh et al. 1997). Salih et al. (1987) reported that treatment affected colostrum selenium with 0.25 mg/kg dietary selenium + 1,500 mg vit E + 5 mg injected selenium and dietary selenium (0.25 mg/kg) alone higher ($P<0.05$) than the control (0.015, 0.049 and 0.031 mg/kg). Also, milk selenium was higher ($P<0.01$) in treatments compared to control (0.0097, 0.0062 and 0.0046 ug/ml). All values are below or close to 0.1 mg/kg required by beef cattle, set by the NRC (1996), so it is risky to rely on milk to provide adequate selenium to calves. If dams are selenium deficient and remain un-supplemented postpartum, parenteral supplementation of newborn beef calves with 1.38 mg intra-muscular. selenium as selenite does not result in normal selenium status 1 month later (Enjalbert et al. 1999).

Lacetera et al. (1996) administered sodium selenite (5 mg/100 kg BW) and vitamin E (alpha-tocopherol, 25 IU/100 kg) to Holstein cows before calving. The cows given selenium and vitamin E produced more colostrum than did their nontreated counterparts at first milking ($P<0.05$) and for the average of 4 milkings ($P<0.005$). Overall production of colostrum was 22 % higher in treated cows (8.8 kg vs 7.2 kg). Milk yield after 4, 8 and 12 wk of lactation was 10 % higher ($P<0.005$) compared to nontreated animals (29 kg vs 26 kg).

Grace et al. (1997) gave two 30 g intraruminal selenium boluses (10 % Se; 90 % Fe by weight) to a group of cows while another similar group was injected subcutaneously with 500 mg selenium as barium selenate. The cows given intraruminal selenium boluses in June before calving in early August had milk selenium values of

107, 100 and 103 nmol/L on days 76, 147 and 190 after treatment, respectively. In the group administered 500 mg selenium as barium selenate subcutaneously, milk selenium concentrations were 95, 117 and 132 nmol/L on days 86, 157 and 265 after treatment, respectively. These values were higher ($P<0.001$) than values in untreated groups.

Placental transfer of selenium has already been demonstrated in cattle because the maternal supplementation of cows in late gestation increases selenium reserves in the liver of the fetus and newborn (Van Saun et al. 1989; Abdelrahman and Kincaid , 1995). Paired dam-fetus serum, whole blood and liver samples were collected from 101 pregnant dairy cows at slaughter by Van Saun et al. (1989) to establish mean values for fetal tissue selenium concentration and to determine relationships between maternal and fetal selenium status. Mean fetal liver selenium concentration was consistently greater ($P<0.05$-$0.001$) than mean maternal liver selenium concentration across gestation, suggesting fetal concentration ability and a possible selenium storage function for the fetal liver. In contrast to liver selenium, mean maternal serum selenium concentration was consistently greater ($P<0.05$-$0.001$) than mean fetal serum selenium until 8.5 mo, when maternal serum selenium declined rapidly. At the same time, maternal liver selenium also declined, and this may be related to losses to colostrum or rapid fetal growth. The improvement in selenium status in calves as a result of maternal supplementation has been found to be greater pre-partum than post-partum, suggesting that placental transfer of selenium is more efficient than milk transfer

(Enjalbert et al. 1999). It has been suggested that organic selenium may well cross the placenta more efficiently than inorganic selenium (Awadeh et al. 1997). Selenate transport across placental membrane vesicles has been described (Boyd and Shennan, 1986; Shennan, 1988) as an anion exchange pathway shared with sulphate, which is sodium independent and inhibited by other tetrahedral anions.

When feeding pelleted alfalfa hay (0.085 mg/kg selenium), soybean meal (0.313 mg/kg selenium) and 90 mg of selenium as sodium selenite/kg in a salt mineral mix ad libitum or pelleted hay plus soybean meal to two groups of 20 month old pregnant Hereford heifers, Koller et al. (1984) found that the pre-suckled colostrum contained a relatively high amount (0.124 mg/L) of selenium in cows from the first group and much lower amounts ($P<0.01$) in the second group (0.070 mg/L) and in the control group (0.032 mg/L) fed only on pelleted alfalfa hay. By the first week after calving these values had decreased markedly, with the milk selenium concentrations of 0.037 for the first group, 0.025 for the second and 0.009 mg/L selenium for the control. There was obvious transport of selenium across the placenta from dam to offspring and, when lower amounts of selenium were available to the mother, a greater proportion was present in the neonate.

After providing selenium to deficient, pregnant beef cows by different methods (group A: control; group B: 1 selenium bolus at day 0 and a second at day 119; group C: 1 selenium bolus at day 0; group D: 2 selenium pellets at day 0), Campbell et al. (1990) reported that all selenium-supplemented cows (groups B, C and D) had higher ($P<0.001$) colostrum selenium than did controls (0.072, 0.052 and 0.060 mg/kg vs 0.017 mg/kg). Calf birth weights were not affected by treatment.

Knowles et al. (1999) supplied selenium as a drench to grazing dairy cows in mid-lactation at a rate of 2 or 4 mg/d of organic or inorganic selenium. These cows were grazing pastures with selenium concentrations of 0.035 mg/kg of DM, which contributed approximately 0.5 mg/d of selenium to all cows. These authors found that the organic selenium (Se-yeast) was 2 to 3 times more effective than sodium selenite at increasing milk selenium concentration, and low intakes of both supplements were 27 % more efficient per mg of selenium administered than were high intakes. Treated cows had 1.5 to 5.5 times more selenium concentration in milk than the control group at day 133. Milk yield was not affected by selenium treatments. Mean selenium concentrations in liver samples taken at the end of the trial were 920, 1930, 2710, 2430 and 3290 nmol of Se/kg of fresh liver weight for control cows, cows treated with low and high concentrations of sodium selenite and cows treated with low and high concentrations of Se-yeast, respectively.

Ortman and Pehrson (1999) fed forty-two dairy cows a basic ration containing 0.1 to 0.12 mg selenium/kg DM for five months and then divided the cows into four groups of 10 or 11, as similar as possible in age and stage of lactation. During the next 3 months the cows in groups 1, 2 and 3 were supplemented with 3 mg selenium daily, while the cows in group 4 remained un-supplemented. In group 1 the supplement was sodium selenite, in group 2 it was sodium selenate and in group 3, it was Se-yeast.

Selenium concentration in plasma increased in all supplemented groups, and reached a plateau within 4 weeks, at 75 ug/L in group 1, 80 ug/L in group 2 and 90 ug/L in group 3. Plasma selenium concentration remained at about 50 ug/L in group 4. Concentrations of selenium in milk during the study period was16.4 ug/L in group 1, 16.4 ug/L in group 2 and 31.2 ug/L in group 3, whereas the concentration of selenium remained at about 14 ug/L in group 4. There was no difference between the two inorganic compounds in raising milk selenium concentrations and these had a very limited effect. Organic selenium was much more effective than inorganic in increasing the concentration of selenium in milk. The concentration of selenium in milk increased in all supplemented groups and reached a plateau value within one week after the start of supplementation. This value was about 130 % higher for the Se-yeast group when compared to the control group, while inorganic selenium had a plateau value about 20 % higher than that of the control.

The hypothesis that the selenium status of sucking calves might be improved if their dams were offered an organic selenium compound instead of sodium selenite was investigated by Pehrson et al. (1999). A commercial Hereford herd of cows, who were maintained on pastures year round and had free access to hay and a vitamin mineral supplement during the winter and also during the spring and summer when most of their nutritional needs were supplied by pasture, was divided into two groups. The mineral supplement used before the start of the trial contained 30 mg selenium/kg as sodium selenite. The control group still had free access to the mineral supplement with sodium selenite. The experimental group had free access to a mineral supplement that also contained 30 mg selenium/kg, but as organic selenium in the form of Se-yeast. Supplementation started on March 15 and blood samples were collected on April 28 and on June 3. For the two collection dates mean plasma selenium concentration (ug/L) in plasma of cows were 66 and 56 for cows on selenite and 65 and 81 for cows on Se-yeast and 36 and 32 for calves from dams on selenite and 47 and 50 for calves from dams on Se-yeast, respectively. Plasma selenium concentrations were not different on the first date but Se-yeast was higher ($P<0.007$) than selenite on the second sampling. Plasma selenium concentrations were higher ($P<0.006$) in the first sampling for Se-yeast as well as on the second date ($P<0.002$). Concentrations (ug/L) of milk selenium were 13.7 and 11.7 for cows on selenite and 17.0 and 17.5 for cows on Se-yeast, respectively. At both samplings, the milk from cows given Se-yeast supplement contained a higher ($P<0.001$) concentration of selenium than milk of of cows given sodium selenite. There were no differences ($P<0.05$) in weight gains of calves at the end of the study. Suckling calves may be at risk of selenium deficiency if their dams are fed on selenium-deficient feedstuffs, even if cows are supplemented with sodium selenite. Use of organic selenium in the form of a yeast would greatly reduce this risk. Selenium in the form of yeast is more efficient than sodium selenite at increasing the selenium status of suckling calves only when the basic feed is deficient or marginal in selenium.

The final 60 days of gestation, which correspond to the dry period, is a critical period for transfer of selenium from the dam to the fetus, which can cause a significant loss to the selenium stores of the peripartum cow (Abdelrahman and Kincaid, 1995). Maternal liver and serum selenium tends to decrease after 8.5 mo gestation, possibly as a result of selenium mobilization for colostrum or rapid fetal growth (Van Saun et al. 1989). Intestinal absorption of selenium is inhibited by excess dietary sulfur (NRC, 1980; Peter et al. 1981; Harrison and Conrad, 1984b), a component of many anionic salts. However, Gant et al.(1998) found that anionic salts can be administered to prevent milk fever without danger of significantly reducing the transfer of selenium from the dam to the calf and without compromising the selenium status of the cow when the salts are provided for 14 to 21 days before calving. Allen and Miller (1981) have shown that in blood $^{75}$Se was at peak concentration 3 to 5 h after an intravenous dose. Peak $^{75}$Se activity in plasma ocurred at 6 h. There was a sharp decline in activity after the first 8 h, until the $^{75}$Se activity of blood and plasma leveled off by days 2 and 3. Initial entry of $^{75}$Se into milk occurred within the first hour after dosing, rising to a peak activity at 3 to 4 h. This was followed by a sharp reduction and then a slow decline over the remainder of the week.

Most selenium in animal tissues is bound to protein and research has shown the presence of numerous selenoproteins other than glutathione peroxidase. One of these is selenoprotein P, which is synthesized mainly in the liver and secreted into plasma. Burk and Hill (1993) found that, in rats, selenoprotein P contains about 65 % of the selenium in the plasma. To determine the distribution in cattle, two groups of growing calves were assigned by Awadeh et al. (1998) to receive either adequate (0.41 ug/g) or excess (0.73 ug/g) dietary selenium as sodium selenite. Proteins in serum collected from the calves were separated into albumin, glutathione peroxidase, and selenoprotein P fractions, and the concentration of selenium in each was determined. Concentration of selenium within serum was elevated by dietary selenium supplementation. The mean percentage distribution of serum selenium among protein fractions was 68.9 % in selenoprotein P, 19.2 % in albumin, and 11.8 % in GSHpx. The selenium distribution pattern in serum of calves was very similar to the distribution pattern found in rats (Deagen et al. 1993) and humans (Xia et al. 1992), indicating the presence of selenoprotein P in cattle.

In a second study, Awadeh et al. (1998) assigned mature cows to receive four experimental salt mixes containing 20, 60, or 120 ug of selenium as sodium selenite/g of salt mix, while the fourth treatment was 60 ug of selenium as selenized yeast/g of salt mix. Cows given salt with 120 ug of selenium as sodium selenite or 60 ug of selenium as selenized yeast had the highest concentrations of selenium in whole blood. However, concentrations of selenium in serum did not differ among treatments.

Using the the method of Whetter and Ullrey (1978), the Michigan State University Animal Health Diagnostic Laboratory expects serum selenium values (nanograms per mililiter) of livestock to increase gradually with age, with values of 50 to 80 for neonates of sheep and cattle, whereas values for adults of these species are in the ranges of 70 to 100 and of 120 to 150 ng/g, respectively. Normal liver selenium concentrations are considered to range between 1.2 and 2.0 ug/g on a dry weight basis, regardless of the species or age.

The NRC (2001) states that "methionine is often cited as being the most limiting amino acid for lactating cows." When there is a low methionine intake, the dietary selenium-methionine is sequestered in protein (Levander and Burk, 1990). On the other hand, a high intake of methionine will lead to catabolism of seleno-methionine, resulting in selenium release. Also, when dietary selenium supply is reduced, turnover of seleno-methionine from tissues continues to provide available selenium to the animal (Levander, 1986; Pehrson, 1993b), thus playing a very important role in selenium homeostasis. Furthermore, in order to conserve selenium for essential seleno-protein synthesis, animals with low dietary selenium intake will minimize urinary excretion (Levander, 1986; Levander and Burk, 1990).

## CHAPTER 3

## SUPPLEMENTING ORGANIC AND INORGANIC SELENIUM ON YEARLING CATTLE PERFORMANCE AND TISSUE SELENIUM CONCENTRATIONS.

### Introduction

It has been well documented that when sodium selenite is given orally to mature ruminants, compared to monogastric species, most of the selenium is excreted in the feces (Paulson et al., 1966; Wright and Bell, 1966; Lopez et al., 1968). This is due, presumably, to microorganisms and the reducing environment within the rumen that convert the selenite to less soluble forms, such as elemental selenium or selenides (Cousins and Cairney, 1961).

Perhson (1993b) reported Se-yeast to be approximately twice as active as selenite for increasing the glutathione peroxidase activity in RBCs of selenium deficient young cattle. Almost one half of the total organic selenium in Se-yeast is in the form of selenomethionine.

Selenium supplemented ewes produced lambs with a weaning weight of 25.2 kg compared to 22.1 kg from an unsupplemented group (Langlands et al.,1991). In another study, Donald et al.(1993) also found that lambs reared by ewes given a selenium supplement grew more rapidly than non-supplemented controls (176 vs. 144 g/day).

Wichtel et al. (1996a) conducted two trials on selenium supplementation of grazing calves (5 months of age) and found positive weight responses in one herd but not in the other. Lacetera et al. (1996) reported no impact of selenium on growth of calves born of selenium supplemented dams.

The objectives of this experiment were: 1) to compare growth rates of Angus yearling calves that were supplemented with organic and inorganic selenium at two levels, and 2) to evaluate treatment effects on liver and plasma selenium concentrations.

## Materials and Methods

Forty Angus yearling calves of both sexes with an average weight of 235 kg for steers and 231 kg for heifers were utilized in a 84 d experiment at the North Florida Research and Education Center, Quincy, Florida. The experiment was conducted from February to May in 1998. The animals were dewormed with Dectomax at 1 ml/ 50 kg before the start of the trial. At the start of the trial, the animals were weighed, randomly allotted to ten 5 x 8 m earth pens in an open barn and blood samples and liver biopsies collected. Afterwards, the animals were weighed every 28 d and blood samples collected. At the end of the 84 d trial, liver biopsy and blood samples were again collected and weights determined. All procedures of this experiment were approved by the University of Florida Animal Care and Use Committee.

Blood was collected via jugular venipuncture with an 18-gauge needle into heparinized vacuum blood collection tubes. Liver biopsy samples were collected with trocar and cannula by the procedure of Chapman et al. (1963) at the 11th intercostal space. Selenium blood and liver sample concentrations were determined using the Whetter and Ullrey (1978) improved fluorometric method.

The cattle were fed an average of 10 kg/d of bahiagrass hay (0.04 mg/kg selenium) and a concentrate, prepared from corn, cottonseed meal and cottonseed hulls, at a rate of 2.5 kg/animal/day. They were also fed ryegrass hay (0.05 mg/kg seleniium) and oat hay (0.04 mg/kg selenium). Five treatments consisted of a control (no selenium in feed) and two levels of selenium in concentrate (0.1 and 0.3 mg/kg) each for the inorganic, sodium selenite, and organic source, Se-yeast (Alltech Biotechnology Center, 3031 Catnip Hill Pike, Nicholasville, Kentucky 40356). The selenium was mixed with the concentrate. Prior to the start of the experiment the calves had access to a free-choice mineral salt with 0.3 mg/kg selenium as sodium selenite, with an average consumption of 40 g/d

The composition of the concentrate feed was as follows::

Table 1. Composition of concentrate feed and its selenium concentrations[a]

| Ingredients | % | Selenium concentration by analysis | Nutrients |
|---|---|---|---|
| Corn (ground) | 75 | 0.15 mg/kg | 10 % protein |
| Cotton seed hulls | 20 | | 1.89 Mcal/kg NEm |
| Cotton seed meal | 4 | 0.30 mg/kg | 1.24 Mcal/kg Neg |
| Salt mineral mix | 1 | | |

[a] Yea-Sacc (organic selenium source) was added at a rate of 272.7 g/ton to provide 0.3 mg/kg selenium. For the 0.1 mg/kg treatment, Yea-Sacc was added at a rate of 90.9 g/ton..

The following mineral mix with no selenium was mixed with the concentrate:

Table 2. Composition of the salt mineral mix that was part of the concentrate feed.

| Ingredient | | % |
|---|---|---|
| Calcium (Ca) | Min | 9.5000 |
| " | Max | 11.0000 |
| Phosphorus (P) | Min | 9.0000 |
| Salt (NaCl) | Min | 30.0000 |
| " | Max | 34.0000 |
| Fluorine (F) | Max | 0.0900 |
| Magnesium (Mg) | Min | 1.1000 |
| Sulfur (S) | Min | 1.8500 |
| Cobalt (Co) | Min | 0.0020 |
| Iodine (I) | Min | 0.0160 |
| Iron (Fe) | Min | 0.5000 |
| Manganese (Mn) | Min | 0.5000 |
| Zinc (Zn) | Min | 1.5000 |

Plasma and liver selenium as well as the weight data were analyzed by repeated measures analysis of variance in a completely randomized design using the GLM procedure of SAS (1996). Treatment means were compared with a LSD test and the experimental unit was each pen with four calves.

## Results and Discussion

### Weight Gains of the Calves

The highest daily gains were for the organic 0.1 mg/kg selenium treatment with 1.91 kg/day (Table 3). This treatment had higher ($P<0.05$) gains than all other

Table 3. Daily gains of Angus calves supplemented with two levels and two sources of selenium (n = 40)[a]

| Treatment | Time of experiment (d) | | |
|---|---|---|---|
| | 0-28 | 29-56 | 57-84 |
| Control | $2.08^{b}$ | $1.24^{b}$ | $1.02^{b}$ |
| 0.1 mg/kg selenite | $2.66^{bc}$ | $1.06^{b}$ | $1.33^{bc}$ |
| 0.3 mg/kg selenite | $2.09^{b}$ | $0.83^{b}$ | $1.51^{bc}$ |
| 0.1 mg/kg Se-yeast | $2.88^{c}$ | $1.06^{b}$ | $1.83^{c}$ |
| 0.3 mg/kg Se-yeast | $2.05^{b}$ | $1.07^{b}$ | $1.96^{c}$ |

[a] Data represent LS means and standard error = 0.24.

[bc] Means with different superscripts within a column differ (P<0.05), by LSD test.

treatments. Several research reports (Langlands et al., 1991; Donald et al.,1993; Wichtel et al.,1994, 1996a) have noted improvement in growth or body weight of ruminants that had received selenium supplementation. Live weights of breeding ewes receiving supplemental selenium (intra-ruminal pellet) were generally greater ($P<0.05$) than those of comparable un-supplemented ewes (Langlands et al., 1991). Donald et al. (1993) reported that lambs born to selenium supplemented (intra-ruminal pellet) dams were 8 % heavier at birth ($P<0.05$) and 19 % heavier at weaning ($P<0.001$), and grew faster ($P<0.01$) than those reared by un-supplemented ewes (176 vs 144 g/day). Supplemental selenium (selenium pellet) enhanced live-weight gains by 26 % during the final month of a 95 day study using Angora goat kids with a 3-4 mo initial age (Wichtel et al., 1996c). The same authors (Wichtel et al., 1996d) found no response to selenium supplementation (0.1 mg/kg selenate) in a 21 day study with Angora goat kids of 6-7 mo of age. The temporal relationships between treatment and plasma thyroxine concentrations indicate that responses should be expected to occur no sooner than 3 wk post-supplementation. In studies with other species, Wichtell et al. (1996a) found no difference in weight gains in one of two herds of calves studied, while Lacetera et al. (1996) also reported no impact of selenium on growth rate of calves born to dams that had received selenium supplementation.

In the present study, inorganic and organic selenium were compared at two levels and organic selenium (0.1 mg/kg) treatment resulted in higher weight gains than other treatments, except in the last 28 d period where the 0.3 mg/kg organic selenium treatment was the highest. With organic selenium sources, seleno-methionine incorporation into protein directly reflects its concentration in the diet. Inorganic selenium (sodium selenite) is only used or incorporated into a specific aminoacid (cysteine) in the selenoproteins (Burk, 1991) and excess absorbed selenium is excreted as trimethyl selenomium or exhaled as dimethyl selenide (Janghorbani et al., 1990). This can explain why the inorganic 0.3 mg/kg treatment was not as effective as the organic 0.1 mg/kg in stimulating higher weight gains. The inorganic selenium was excreted or exhaled. However, this does not explain why the organic 0.3 mg/kg treatment did not stimulate the highest weight gains. Excess selenium from the organic 0.3 mg/kg treatment was stored in liver at higher concentrations than the other treatments (Table 5). This could mean that organic sources at this concentration would be stored in liver and could be used when dietary deficiencies occur.

The selenium concentration of the forages and other feeds consumed by the Angus calves in this study was variable. Prior to the experiment the animals were grazing bahiagrass and bermuda grass pastures with selenium concentrations of 0.012 to 0.042 mg/kg and the hays provided during the experiment had values of 0.05 mg/kg for the ryegrass hay and 0.04 for the oat hay. The selenium levels for the control concentrate were 0.05 mg/kg. These levels are not adequate but also are not highly deficient. All calves also had access to a mineral supplement which contained 0.3 mg/kg selenium as sodium selenite before the trial began, which would allow animals to have adequate storage. This could have negated any effect of the treatments on weight gains of the calves. Pehrson et al. (1999) have reported that selenium in the form of yeast is more efficient than sodium selenite only when the basic feed is deficient or marginal in selenium. In this case the calves had been consuming a mineral supplement with selenium. Although the grass or hay was selenium deficient, their selenium stores were adequate.

### Plasma Selenium Concentrations

Plasma selenium differed ($P<0.05$) among treatments (Table 4). Plasma selenium concentrations in calves at the start of the study were not highly deficient with the control being highest (0.06 mg/kg) but decreasing most at the end (0.028 mg/kg). However, the levels of selenium in the other treatments remained the same (0.1 mg/kg selenite) or increased ( 0.3 mg/kg selenite and 0.1 and 0.3 mg/kg Se-yeast) in the first 28 days and then tended to decrease, with the exception of the 0.3 mg/kg organic treatment where the trend was to increase with time.

The decrease in the inorganic selenium could be attributed to the fact that functional ruminants tend to excrete orally administered sodium selenite (Paulsen et al., 1966; Wright and Bell, 1966; Lopez et al., 1968). In the present study, where initial selenium stores were adequate, any supplemental inorganic selenium could have had lower absorption or excretion, thus presenting lower values in plasma at the end of the experiment. Based on selenium concentrations in serum, skeletal muscle and liver of lambs and feeder cattle (Ullrey et al., 1977), supplemental selenium as sodium selenite was not retained or absorbed as efficiently as organic forms.

Ullrey (1987) reported a value of 0.08 ug/mL of serum selenium as indicator of an adequate selenium status. With this in mind, Podoll et al. (1992) reported that

Table 4. Plasma selenium concentrations (ug/ml) in Angus calves supplemented with two levels and two sources of selenium (n = 40)$^{a}$

| Treatment | Days of experiment | | | |
|---|---|---|---|---|
| | 0 | 28 | 56 | 84 |
| Control | 0.061$^{b}$ | 0.037$^{b}$ | 0.034$^{b}$ | 0.028$^{b}$ |
| 0.1 mg/kg (selenite) | 0.048$^{c}$ | 0.047$^{bd}$ | 0.044$^{bc}$ | 0.028$^{b}$ |
| 0.3 mg/kg (selenite) | 0.053$^{bc}$ | 0.059$^{c}$ | 0.050$^{c}$ | 0.040$^{c}$ |
| 0.1 mg/kg (Se-yeast) | 0.050$^{c}$ | 0.056$^{cd}$ | 0.046$^{c}$ | 0.034$^{bc}$ |
| 0.3 mg/kg (Se-yeast) | 0.045$^{c}$ | 0.052$^{cd}$ | 0.065$^{d}$ | 0.058$^{d}$ |

[a] Data represent LS means and standard error = 0.003.

[bcd] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

with a DMI of 24 kg/day with an analyzed selenium concentration of 0.3 mg/kg of DM, 7.2 mg of selenium was consumed per day to reach this concentration.

The concentration of selenium in plasma was significantly higher ($P<0.01$) in dairy heifers fed 2 mg selenium/d as Se-yeast (1100 nmol/L) than in heifers fed the same amount of selenium from sodium selenite or sodium selenate (900 nmol/L). Plasma selenium concentration increased in all the supplemented groups and reached a plateau after 4 wks in the sodium selenate and Se-yeast heifers and after 6 wks in the sodium selenite group (Ortman et al., 1999).

In the present study the selenium concentration in the concentrate was balanced to provide a high of 0.3 mg/kg and consumed at 2.5 kg per calf, so an intake of 0.75 mg/d of selenium came from the feed plus 0.5 mg/d from the hay (from an estimated 10 kg daily consumption). This totals 1.25 mg of selenium consumed per day. It is thus clear that selenium intake was lower in our study as compared to others. However, efficiency of organic sources was manifested in higher plasma selenium concentrations than the inorganic sources, although adequate selenium status as indicated by Ullrey (1987) was never reached.

### Liver Selenium Concentrations

All liver selenium concentrations were above the critical levels (0.03 mg/kg). The 0.3 mg/kg Se-yeast treatment was higher ($P<0.05$) than all other treatments in liver selenium after 84 d. Animals receiving 0.3 mg/kg selenium as selenite had higher liver selenium than the control or 0.1 mg/kg selenite treatments (Table 5).

Table 5. Liver selenium concentrations (ug/g) of Angus cows supplemented with two levels and two sources of selenium (n = 40)[a]

| Treatment | Time of experiment | |
|---|---|---|
| | Day 0 | Day 84 |
| Control | $0.79^{b}$ | $0.57^{b}$ |
| 0.1 mg/kg (selenite) | $0.66^{bc}$ | $0.64^{bc}$ |
| 0.3 mg/kg (selenite) | $0.73^{bc}$ | $0.82^{d}$ |
| 0.1 mg/kg (Se-yeast) | $0.58^{c}$ | $0.77^{cd}$ |
| 0.3 mg/kg (Se-yeast) | $0.72^{bc}$ | $1.09^{e}$ |

[a] Data represent LS means and standard error = 0.049.

[bcd] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

The control treatment decreased in liver selenium concentrations with time, and this would be normal as animals would be depleting selenium liver storage when not receiving sufficient selenium supplementation. Considering the high weight gains of control calves, and positive effects reported by Langlands et al. (1991), Donald et al. (1993), and Wichtell et al. (1994, 1996a) of selenium supplementation on performance of ruminants, it is very possible that depletion of liver selenium storage in control calves could be due to use of selenium for growth, mediated through thyroid hormone metabolism (Beckett et al.,1987; Beckett et al., 1989; Arthur et al., 1987; Arthur and Beckett, 1989).

The 0.1 mg/kg selenite treatment tended to maintain similar levels of selenium in liver at the end of the experiment (0.64 mg/kg) compared to the levels at the start (0.66 mg/kg). It is known that selenite is reduced to selenide (Ganther, 1979) and then methylated to form dimethylselenide, which is exhaled through the lungs, or trimethyl-selenomium which is excreted via the kidney (Levander, 1986; Janghorbani et al., 1990). Also, in the rumen, selenite is converted to selenide, which as stated before is not absorbed. Liver selenium concentration was not higher possibly because of the initial adequate liver concentrations.

The initial liver selenium concentrations were higher in the 0.3 mg/kg selenite treated calves and in the 0.3 mg/kg Se-yeast group than the 0.1 mg/kg selenite and 0.1 mg/kg Se-yeast treated calves. However, the control calves had the highest initial selenium concentrations in liver although they decreased with time. All treatments increased liver selenium concentrations from day 0 to day 84, except the 0.1 mg/kg selenite group which decreased slightly (0.66 to 0.64 mg/kg). It is possible that all this selenium was not excreted but was stored in the liver. If the metabolic pathways of inorganic selenium are considered (Paulsen et al., 1966; Wright and Bell, 1966; Lopez et al., 1968), this should not have been the case, but the rumen microbial population is capable of converting a significant portion of selenide to organic forms which can be actively absorbed (Mahan, 1994; Mahan, 1995). A high carbohydrate diet also favors a higher conversion of inorganic selenium to organic forms in the rumen (Pehrson, 1993b). This could have increased liver selenium concentrations for the 0.3 mg/kg selenite treatment.

In the mid-1970's, metabolic studies indicated that selenium-methionine (Se-Met) is absorbed and retained well, suggesting its use for nutritional selenium supplementation (Griffiths et al., 1976). At about the same time, high Se-yeast was introduced as a very economical food source of Se-Met. Se-Met is incorporated into body proteins allowing selenium to be stored in the organism and reversibly released by normal metabolic processes, thus offering an advantage over other selenium compounds (Schrauzer, 2000). Ingested Se-Met is absorbed in the small intestine via the $Na^+$-dependent neutral amino acid transport system (Vendeland et al., 1994). Any Se-Met that is not immediately metabolized is incorporated into organs with high rates of protein synthesis, such as the skeletal muscles, erythrocytes, pancreas, liver, kidney, stomach and the gastrointestinal mucosa (Schrauzer, 2000). Se-Met is also significantly retained by the brain, indicating that it is an active form of selenium for incorporation into brain proteins (Gronbaek and Thorlacius-Ussing, 1992). Se-Met supplemented animals maintained higher activities of selenoenzymes during selenium depletion for longer periods than previously selenite-supplemented animals. So, it would appear that in the present study the Se-yeast supplemented animals stored more selenium in organs such as liver, than did the selenite supplemented ones.

In sheep supplemented with either 3, 6 or 9 mg/kg Se (as-fed basis) for 10, 20 or 30 d, Echeverria et al. (1988) found that liver selenium concentration increased ($P<0.01$) from 0.85 to an average of 8.30 mg/kg Se as supplemental selenium increased, and liver selenium from sheep fed control diets was lower ($P<0.01$) than that of sheep fed added selenium. Sheep fed for 30 d accumulated more ($P<0.01$) selenium in liver than those fed for 10 or 20 d. Serum selenium increased ($P<0.01$) from 0.09 ug/ml in the un-supplemented controls to 0.25 ug/ml for sheep fed 9 mg/kg added selenium.

The increase in liver selenium concentrations in calves treated with 0.1 ppm Se-yeast can also be explained considering that the metabolic pathway for organic selenium is different than that of the inorganic form. Organic selenium is present as seleno-methionine and this compound is nonspecifically incorporated as such into all proteins as a substitute for methionine (Gerloff, 1992), which likely accounts for the tendency of this form of selenium to be retained in the body (Griffiths et al., 1976). The stored form of selenium is in a non-functional state, i.e. not used immediately for the formation of biologically functional selenoproteins (Mahan, 1994; Mahan, 1995).

Calves on the 0.3 ppm Se-yeast treatment accumulated the highest liver selenium concentrations in the treated calves. Selenium provided in the form of yeast had a

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higher bioavailability than the inorganic compounds (Mahan, 1995). It is also better retained in the body (Griffiths et al., 1976). Selenium can be absorbed in an organic form as a component of amino acids (seleno-methionine) by an active transport mechanism (Pehrson, 1993b).

## Summary

In an 84 day experiment, Angus calves were supplemented in the concentrate mixture with organic and inorganic selenium at two levels each (0.1 and 0.3 ppm) for 84 days. There was a difference ($P<0.05$) among treatments with the organic 0.1 mg/kg treatment resulting in higher weight gains (161 kg) than all other treatments. There were no differences ($P>0.05$) among the other 4 treatments. There were differences ($P>0.05$) among treatments in plasma selenium concentrations, with the 0.3 mg/kg Se-yeast giving the highest values, followed by the 0.3 mg/kg selenite, then the 0.1 mg/kg Se-yeast, while the 0.1 mg/kg selenite and the control had the lowest but similar values. Plasma selenium concentrations of control animals tended to decrease with time. The selenium treatments tended to increase plasma concentrations at 28 days and then started to decrease slightly. The 0.3 mg/kg Se-yeast treatment, however, showed less tendency to decline with time. At no time were plasma selenium concentrations adequate. Liver selenium concentrations differed ($P<0.05$) among treatments, with the highest level of supplemental organic selenium (0.3 mg/kg) higher than all other treatments. Animals receiving 0.3 mg/kg selenium as selenite had higher liver selenium than the control or 0.1 mg/kg selenite treatments.

## Implications

The trend in this study is for the organic selenium sources to be more effective in stimulating weight gains and liver selenium concentrations than the inorganic sources. Organic selenium at 0.1 mg/kg stimulated the highest weight gains while organic selenium at 0.3 mg/kg resulted in the highest liver selenium concentrations. There were differences in serum selenium concentrations attributed to selenium source, with organic selenium sources providing higher concentrations, although none of the treatments reached adequate levels in the serum.

# CHAPTER 4
# EFFECT OF SELENIUM SUPPLEMENTATION ON THYROID HORMONE OF GROWING BEEF CALVES

## Introduction

Impaired thyroid hormone metabolism may be responsible for decreased growth and resistance to cold stress in selenium-deficient animals. Some of the consequences of selenium deficiency may result from thyroid changes since iodothyronine 5'-deiodinases (selenium dependant enzymes) are responsible for the production of 3,5,3' -triiodothyronine, the active form of the hormone.

In selenium-deficient cattle, plasma concentrations of the thyroxine prohormone ($T_4$) increase and those of its active metabolite 3,5,3'-triiodothyronine ($T_3$), decrease (Beckett et al., 1989; Arthur et al., 1990). Consumption of a selenium deficient diet for 23 weeks by Friesian steers had an increasing effect on plasma $T_4$ concentrations, which were higher in selenium deficient animals (62 vs 50 %, respectively). The selenium sufficient group of calves was supplemented with 0.1 mg/kg of diet as sodium selenite (Arthur et al., 1988).

Wichtel et al. (1996a) reported that selenium supplementation increased plasma $T_3$ concentration and decreased plasma $T_4$ of Holstein calves in two herds. This has also been demonstrated in other species. It has been reported that selenium deficiency in chicks decreased circulating $T_4$ and $T_3$ concentrations whereas selenium supplementation increased the plasma concentration of $T_4$ and $T_3$ (Jensen et al., 1986).

Guo et al.(1998) concluded that thyroid hormone metabolism can surely be regulated by selenium through iodothyronine 5'-deiodinase. Selenium deficiency in rats decreased conversion of $T_4$ to $T_3$ in liver homogenates and in plasma (Beckett et al., 1987; Arthur, 1991).

The objective of this experiment was to test the hypothesis that supplementation with selenium alters the plasma concentration of $T_4$ and $T_3$ and to determine if the source of selenium, inorganic or organic, has an effect.

## Materials and Methods

Twenty Angus yearling calves from a total of 40 animals that had been previously allotted to two sources (organic and inorganic) and two levels of selenium (0.1 and 0.3 mg/kg) were transported from the University of Florida facilities at Quincy to the Dairy Research Unit at Hague, near Gainesville, and subjected to a hay only diet that was low in selenium (0.03 mg/kg).

The twenty Angus yearling calves had finished the previous experiment with average weights of 284, 294, 292, 304 and 301 kg for the control (0.05 mg/kg selenium), 0.3 mg/kg selenite, 0.3 mg/kg Se-yeast, 0.1 mg/kg selenite and 0.1 mg/kg Se-yeast (Alltech Biotechnology Center, Nicholasville, KY) levels in the concentrate, which was fed at a rate of 2.5 kg per animal/day and they had ad libitum access to bahiagrass hay (0.042 mg/kg selenium) during 84 days.

Upon arrival at the Dairy Research Unit, the calves were tethered inside a barn in individual stalls on rubber matting and allowed 8 days to adapt to the hay diet (0.03 mg/kg selenium) as sole feed, with continuous access to water. The adaptation to the diet was assessed by weighing orts and there were none after two days.

On day 9 feed was withheld and on day 10 thyrothropin-releasing-hormone (pGlu-His-Pro Amide acetate salt min 97 %) from Sigma (P.O. Box 14508, St. Louis, Mo 63178) was diluted in saline to a concentration of 10 ug/ml and administered intravenously at a dose of 0.33 ug/kg of BW. Blood samples were collected according to the following schedule: -30, 0, 30, 60, 90, 180, 300, 480 and 720 minutes relative to the TRH administration at time zero.

The blood samples were collected by jugular venipuncture into citrate anticoagulant tubes (Vacutainers, Becton Dickinson, Franklin Lakes, NJ) and placed on ice. They were centrifuged within one hour and the plasma was frozen at -20 C for the thyroid hormone assays. Blood assays for $T_3$ and $T_4$ were performed using Diagnostic Products Corporation (Los Angeles, CA) Total $T_3$ Coat-A-Count radioimmuno assay kits, TKT35 and TKC45, respectively. The assay sensitivity for $T_3$ was 0.07 ng/ml while for $T_4$ it was 1.6 ng/ml as determined in the University of Florida's Biotechnology Developmental Institute (12085 Research Dr., Alachua, Fl 32615).

The data were analyzed as a repeated measures experiment in a completely randomized design following the GLM procedure of SAS (1996) and comparing the means by an LSD test.

## Results and Discussion

The concentrations of $T_3$ in plasma of Angus calves before and after thyrotropin-releasing hormone are shown in Table 6. There were differences among treatments at 0, 30, 90, 180, and 720 min, with the 0.1 mg/kg selenite treatment having the highest ($P<0.05$) $T_3$ concentrations (0.96 - 1.43 ng/ml). At 60, 300 and 480 min after the challenge, no differences ($P<0.05$) were found among treatments. Interactions between selenium concentrations and time of collection after the challenge was not apparent for any variable. Plasma $T_3$ concentrations were affected ($P<0.05$) by collection times.

In selenium deficient animals, $T_3$ plasma concentration tended to decrease with time whereas in selenium sufficient animals these values increased, as reported by Arthur et al. (1988). This occurs because when there is adequate selenium in the diet, the selenium dependant enzyme iodothyronine-5'-deiodinase can convert $T_4$ (L-thyroxine) to its more active form, 3,5,3'- triiodo-L-thyronine ($T_3$), and therefore this hormone increases in plasma. Wichtel et al. (1996a) reported that supplementation of calves with intraruminal selenium pellets increased the basal plasma concentration of $T_3$ (0.16 to 0.20 ng/ml). In our study, the T3 concentrations started at 1.23 ng/ml and finished with 1.34 ng/ml which agrees with the studies of Arthur et al. (1988) and Wichtel et al. (1996a).

Ortman et al. (1999) reported that the concentration of $T_4$ was significantly higher in an un-supplemented control group, but there were no differences between the supplemented groups (2 mg selenium daily from selenite, selenate and Se-yeast).

Table 6. Effect of selenium sources on plasma T$_3$ concentration (ng/ml) after intravenous thyrotropin releasing hormone challenge in Angus calves (n = 20) [a]

| | Minutes after challenge | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Treatment | 0 | 30 | 60 | 90 | 180 | 300 | 480 | 720 |
| Control | $1.22^{bc}$ | $1.22^{b}$ | $0.84^{b}$ | $1.15^{b}$ | $1.18^{bc}$ | $1.30^{b}$ | $1.13^{b}$ | $1.12^{b}$ |
| 0.1 mg/kg selenite | $1.43^{b}$ | $1.23^{b}$ | $0.96^{b}$ | $1.31^{b}$ | $1.38^{c}$ | $1.18^{b}$ | $1.15^{b}$ | $1.38^{bc}$ |
| 0.3 mg/kg selenite | $1.23^{bc}$ | $0.95^{bc}$ | $0.96^{b}$ | $1.18^{b}$ | $1.03^{b}$ | $1.25^{b}$ | $1.05^{b}$ | $1.23^{bc}$ |
| 0.1 mg/kg yeast | $1.04^{c}$ | $0.87^{c}$ | $0.84^{b}$ | $0.71^{c}$ | $1.16^{bc}$ | $1.17^{b}$ | $0.98^{b}$ | $1.29^{bc}$ |
| 0.3 mg/kg yeast | $1.24^{bc}$ | $1.08^{bc}$ | $0.87^{b}$ | $1.13^{b}$ | $1.03^{b}$ | $1.15^{b}$ | $1.19^{b}$ | $1.47^{c}$ |

[a] Data represent LS means and standard error = 0.11.

[bc] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

In contrast, the $T_3$ concentrations were very similar in all 4 groups. It is possible that there are compensatory systems which actively maintain the concentration of the more metabolically active hormone $T_3$ nearly constant. For example, the activity of the enzyme iodothyronine deiodinase is actually induced in selenium-deficient animals, despite it being a selenium-containing enzyme, and moreover, the degradation of $T_3$ to $T_2$ becomes inhibited when the supply of selenium is limited (Arthur, 1993). This suggests that animals in the Ortman et al. (1999) study were marginally deficient, rather than severely deficient in selenium.

Ullrey et al. (1977) reported that in sheep and beef cattle, tissue selenium levels were higher from diets consisting of natural sources as compared to levels that result from those fortified with selenite. Organic selenium is more available for use by iodothyronine-5'-deiodinase, which is essential for activating the thyroid hormone $T_3$.

In Finland, supplementation with selenium-rich wheat or yeast elevated plasma selenium levels in humans more efficiently than did supplementation with sodium selenate (Levander, 1986) and when supplement was discontinued, the subjects that had been given the organic selenium sources had higher plasma concentrations some weeks later than the group that had been given the inorganic selenium source.

The above results differ from those found in this study. The $T_3$ concentrations in the plasma of Angus calves indicated that there were no effects of the selenium source or level on thyroid hormone metabolism. This could be influenced by the fact that in our study the thyroid hormone concentrations were assessed after a 12 week period of feeding the penned calves hay and a concentrate that contained the different levels and sources of selenium whereas Wichtel et al. (1996b) had administered two intra-ruminal pellets to each calf and the blood samples were collected at 9 weeks. Additionally, the animals were grazing ryegrass and white clover pastures which had levels of 0.02 and 0.03 mg of Se/kg of DM. For the present study, Se levels were of 0.05 mg/kg for the control feed and of 0.012 and 0.042 mg/kg for the pasture and hay, respectively. The somewhat higher dietary selenium in the present study could have negated a source effect. Arthur et al.(1988) found that plasma $T_4$ concentrations at 20 and 23 weeks were higher, respectively, by 50 and 62 % in selenium-deficient steers while the plasma $T_3$ was decreased by 30 and 35 %. Also, Table 6 shows a tendency for $T_3$ levels to increase from 30 to 720 minutes while the control is decreasing. So, it could be possible that the response to a TRH challenge can manifest itself differently in beef cattle due to a breed effect. Bass et al. (2000) report findings that suggest a breed-related difference between Holsteins and Jerseys in selenium metabolism, similar to that reported for Cu, Fe and Zn (Du et al., 1995). However, the reason for lack of response most likely relates to more adequate dietary selenium concentrations compared to other trials. The Angus yearling calves used on this study had been on a mineral supplement which contained adequate selenium until the twelve week study started (Chapter 3).

$T_4$ concentrations are shown in Table 7. There was no statistical difference ($P<0.05$ ) among treatments at the start (0 min), at 60 min and at the end of the experiment (720 min). There were differences ($P<0.05$) among the 30, 90, 180,

Table 7. Effect of selenium sources on $T_4$ response (ng/ml) to thyrotropin release hormone challenge in Angus calves (n = 20)[a]

| Treatment | Minutes after challenge | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 0 | 30 | 60 | 90 | 180 | 300 | 480 | 720 |
| Control | $58.4^{b}$ | $57.9^{b}$ | $44.1^{b}$ | $52.8^{b}$ | $61.0^{b}$ | $62.7^{b}$ | $53.7^{b}$ | $49.9^{b}$ |
| 0.1 mg/kg selenite | $52.5^{b}$ | $46.8^{c}$ | $44.5^{b}$ | $51.2^{b}$ | $59.1^{bc}$ | $47.1^{d}$ | $47.7^{bc}$ | $49.9^{b}$ |
| 0.3 mg/kg selenite | $49.1^{b}$ | $43.6^{c}$ | $40.1^{b}$ | $52.8^{b}$ | $50.9^{c}$ | $50.8^{cd}$ | $43.9^{c}$ | $49.3^{b}$ |
| 0.1 mg/kg yeast | $49.8^{b}$ | $38.0^{c}$ | $39.9^{b}$ | $35.9^{c}$ | $50.4^{c}$ | $55.2^{bc}$ | $49.1^{bc}$ | $47.8^{b}$ |
| 0.3 mg/kg yeast | $52.1^{b}$ | $45.2^{c}$ | $41.4^{b}$ | $49.8^{b}$ | $50.9^{c}$ | $56.9^{bc}$ | $52.3^{b}$ | $55.5^{b}$ |

[a] Data represent LS means and standard error = 3.3.

[bcd] Means with different superscripts within a column differ (P<0.05), by LSD test.

300 and 480 min treatments. However, this has no meaning because the values of these treatments were generally below the control.

Selenium supplementation of calves by Wichtel et al. (1996a) reduced the plasma concentration of $T_4$ from 7.14 to 6.23 ng/ml. Reductions in plasma T4 concentrations have also been reported by Arthur et al. (1988). In this study, the supplementation of selenium in the feed had no effect on the thyroid hormones.

### Summary

The effects of previous selenium supplementation with organic and inorganic sources on thyrotropin-releasing hormone challenge was examined in growing Angus calves. The concentrations of 3,5,3'-triiodo-L-thyronine ($T_3$) and its source, L-thyroxine ($T_4$), were determined in plasma of the calves. Selenium containing deiodinases are responsible for the deiodination of $T_4$ and its conversion to the more active form, $T_3$ . Twenty yearling calves which had been supplemented with inorganic selenium (sodium selenite) and organic (Se-yeast) sources of selenium at two levels each (0.1 and 0.3 ppm) were tethered individually and fed hay for a week before being injected intravenously with thyrotropin-releasing hormone. Blood was collected 30 min before the challenge, at the challenge and then 30, 60, 90, 180, 480 and 720 min after the challenge. There was no apparent effect of selenium sources or levels on $T_3$ concentrations. Differences ($P<0.05$) were found among $T_3$ treatments but the average values were not higher than the control. Interaction between selenium levels and times of collection after the challenge was not apparent for any variable. Statistical significance ($P<0.05$) was found among collection times. There were differences ($P<0.05$) among $T_4$ treatments at 30, 90, 180 and 480 min after challenge but not at 0, 60 and 720 min. However, these values were all below the control values and tended to decline after the challenge.

## Implications

Supplementation with selenium did not alter the plasma concentration of $T_3$ and $T_4$ in yearling Angus calves and the inorganic or organic selenium sources had no apparent effect on thyroid hormone metabolism.

# CHAPTER 5
# EFFECTS OF SUPPLEMENTING SELENIUM TO A BEEF CATTLE COW-CALF HERD ON TISSUE SELENIUM CONCENTRATION

## Introduction

Selenium is an essential nutrient for cattle as selenium deficiency has been associated with nutritional myodegeneration, retained placenta, infertility, abortions, birth of premature, weak or dead calves, decreased immune response and "ill thrift" (Corah, 1991; Maas, 1983; McDowell, 1997). Selenium at high levels is toxic, and these toxic levels are about 100 times the required levels (Oldfield, 1998). Selenium supplementation for cattle was approved in 1978 allowing up to 0.1 mg/kg (FDA, 1978). An increase in the maximum level of supplemental selenium from 0.1 to 0.3 mg/kg in feeds of major food producing animals was approved later (FDA, 1987a,b).

Different maximum tolerance levels have been proposed for selenium, ranging from 2 mg/kg (NRC, 1980) to 4 or 5 mg/kg (Underwood, 1977). With 5 mg/kg of dietary selenium, signs of toxicity require weeks or even months to appear. Several investigators (Smith and Conrad, 1987; Stowe et al., 1988; Podoll et al., 1992) have suggested that selenium intakes for lactating dairy cows should be 5 to 7 mg/d, to support adequate serum selenium concentrations. Ellis et al., (1997) fed healthy adult Holstein cows sodium selenite to provide 0, 3, 20 and 50 mg of selenium/ cow/d (groups A, B, C and D) for 90 days. Serum selenium concentrations in groups receiving 20 or 50 mg/cow/d increased over time ($P<0.01$), compared with the control. Increasing supplemental selenium intake to 100 mg/cow/d further increased serum concentrations ($P<0.05$) from 120 ng/ml to 270 ng/ml. Liver selenium concentration increased in cows of groups C and D, compared with those of group A ($P<0.01$), but was not different between groups A and B. It appears that dietary intake of selenium as sodium selenite as high as 50 to 100 mg/d can be well tolerated by dairy cattle for several weeks or more without adverse clinical consequences.

Though there may be controversy over what is an adequate level of selenium for domestic animals, its essential nutrient status is undeniable because of its many beneficial effects. It has been established that selenium has activity as an antioxidant, in growth, in resistance to cold, in thyroid metabolism, and in immunity (Boyne and Arthur, 1986; Turner and Finch, 1991; Levander et al., 1995).

A two-year experiment was conducted with a beef cow-calf herd to compare methods of selenium supplementation. Treatments included comparison of a short term (Mu-Se) acting source of selenium (sodium selenite) and a long term (Deposel) acting source (barium selenate) of the element. A third treatment utilized an organic form of selenium (Se-yeast). Criteria of comparisons involved selenium concentration of blood, liver, milk and colostrum.

## Materials and Methods

Seventy-five Angus cows (511 kg average weight, and 5-8 mo pregnant as determined by palpation) were utilized in a two year experiment at the Beef

Demonstration Unit of the University of Florida at Chipley, Florida. The cows had BCS ranging from 4 to 7. The duration of the experiment was from December 1996 to December 1998. At the start of the trial, animals were randomly allotted into five groups and blood samples and liver biopsies were collected. Every six months blood samples and liver biopsies were collected for a total of five collections. Colostrum and milk samples from cows in both years were also collected to determine selenium concentrations. All procedures of this experiment were approved by the Inter-institutional Animal Care and Use Committee of the University of Florida.

Blood was collected via jugular venipuncture with an 18-gauge needle into heparinized vacuum blood collection tubes. Liver biopsy samples were collected with trocar and cannula by the procedure of Chapman et al. (1963) at the 11th intercostal space. Colostrum and later produced milk were collected on days 1, 60, 120 and 180 post-partum for each year. Selenium blood, liver, and milk sample concentrations were determined using the Whetter and Ullrey (1978) fluorometric method.

The cows were provided bahiagrass and bermuda pastures (0.05 mg/kg selenium) and grazed ryegrass (0.045 mg/kg selenium) during the winter, and were also fed bahiagrass hay (0.04 mg/kg selenium) during this period. Pastures were sampled once in winter (ryegrass) and once in summer (bermuda and bahiagrass) of each year. Samples were collected at random in a zigzag pattern with the number of samples varying according to the size of the enclosure.

Four treatments consisted of the following: 1) a control (no selenium supplementation), 2) subcutaneous injection of 5 ml of Mu-Se (5 mg selenium per ml sodium selenite, from label, Burns Biotech Labs, Inc. Oakland, CA 94621) every 6 mo, 3) subcutaneous injection of 9 ml of Deposel (50 mg selenium per ml as barium selenate, from label, Grampian Pharmaceuticals Ltd, Leyland.Lancashire PR5 3QN, U.K.) administered only at the initiation of the experiment, and, 4) use of mineral mix with organic selenium (Se-yeast: Yea-Sacc, Alltech Biotech Center, 3031 Catnip Hill Pike, Nicholasville, KY 49356) administered to two groups as a replicate. The free-choice mineral mixture contained 59.99 g/kg Se-yeast (calculated), which resulted in a selenium concentration of 30 mg/kg in the mineral mixture. For both free-choice mixtures the average daily selenium intake was 2.1 mg per cow. Each of the five groups consisted of 15 cows which had been randomly allotted, and these cows remained in the experiment for the two year duration.

The data were analyzed as a repeated measures model in a completely randomized design using the GLM of SAS (1966) and the means compared by an LSD test. The experimental unit was each individual cow.

## Results and Discussion

### Cow Plasma Selenium

Cow plasma selenium concentrations at the start of the experiment were not different ($P<0.05$) among treatments (Table 8) and were similar to data of Ortman and Pehrson (1999) who reported that cows with no selenium supplementation

Table 8. Plasma selenium concentration (mg/L) of Angus cows under different methods and sources of selenium supplementation (n = 75)[a]

| | Months of experiment | | | | |
|---|---|---|---|---|---|
| Sources | 0 | 6 | 12 | 18 | 24 |
| Control | $0.06^{b}$ | $0.03^{b}$ | $0.02^{b}$ | $0.03^{b}$ | $0.02^{b}$ |
| Sodium selenite[1] (Mu-Se) | $0.06^{b}$ | $0.04^{b}$ | $0.05^{c}$ | $0.03^{b}$ | $0.05^{c}$ |
| Barium selenate[2] (Deposel) | $0.06^{b}$ | $0.07^{c}$ | $0.06^{c}$ | $0.04^{bc}$ | $0.06^{cd}$ |
| Free-choice[3] mineral # 1 (Se-yeast) | $0.06^{b}$ | $0.08^{c}$ | $0.06^{c}$ | $0.04^{bc}$ | $0.07^{d}$ |
| Free-choice[3] mineral # 2 (Se-yeast) | $0.07^{b}$ | $0.10^{d}$ | $0.08^{d}$ | $0.05^{c}$ | $0.07^{d}$ |

[a] Data represent LS means and standard error = 0.015.

[bcd] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

[1] Animals received a subcutaneous injection of 5 ml of Mu-Se (Burns Biotech Lab, Inc; 5 mg selenium per ml from sodium selenite) every 6 mo.

[2] Animals received a subcutaneous injection of 9 ml of Deposel (Grampian Pharmaceuticals, Ltd.; 50 mg selenium per ml as barium selenate) administered once.

[3] Animals consummed a free-choice mineral mixture with organic selenium (Se-yeast: Yea-Sacc, Alltech) at a rate of 54.5 g/kg of mineral mix.

maintained an average plasma concentration of 0.05 mg/L. The plasma selenium concentrations of these cows were below 0.07-0.10 mg/L, a level considered as a fairly reliable indicator of selenium adequacy (Gerloff, 1992). McDowell et al. (1984) suggest a critical level for plasma selenium to be 0.03 mg/L. Ellis et al. (1997) indicated that normal serum ranges are from 50 to 100 ug/ml. Acevedo-Maldonado et al. (1999) reported that at calving, the plasma selenium concentrations of Holstein cows were 0.114, 0.125, 0.123 and 0.108 mg/L selenium, respectively, when treated with injectable Mu-Se (1.1 ml/cow) at 30 and 60 d prepartum and with a slow release intraruminal bolus (DuraSe-120) at 60 and 120 d prepartum and these values differed ($P<0.019$) from the control value (0.09 mg/L). At 120 days postpartum, the concentrations were 0.102, 0.108, 0.125 and 0.85 mg/L, respectively. These concentrations were not different ($P<0.019$) than the control (0.107 mg/kg).The pretreatment plasma selenium concentrations of cows in the present study are similar (0.06 to 0.07 mg/L) to those reported by Acevedo-Maldonado et al. (1999), which ranged from 0.060 to 0.078 mg/L. Similar values were found in the present study for the two free-choice mineral mixtures at 180 d when compared to the plasma selenium concentrations found by Acevedo-Maldonado (1999) at 120 d postpartum.

Cows fed 0, 3, 20 or 50 mg/d selenium (Ellis et al., 1997) resulted in serum selenium concentrations of 0.050, 0.065, 0.075, and 0.13 mg/L, respectively. Adequate ranges in plasma for selenium are suggested to be from 0.050 to 0.100 mg/L, so at a dosage of 50 mg/d selenium plasma selenium concentrations would be out of the reference range and would not be a sensitive indicator of supranutritional amounts of selenium intake. At the end of 90 d of treatment, liver selenium concentration in cows of 20 (4.3 ug/g selenium) or 50 mg/d (6.8 ug/g selenium) was 4.3 and 6.8 times greater, respectively, than cows in the control group (1.0 ug/g selenium). In cattle, it appears that liver is a more sensitive indicator of selenium intake above requirements than is serum.

Six months later, differences ($P<0.05$) among treatments were observed, with the control being numerically less than all groups and statistically less than the Deposel and the two free-choice mineral treatments. The Mu-Se treatment animals contained less ($P<0.05$) plasma selenium than the Deposel and the two free-choice mineral mixture treatment cows. The selenium concentrations for all animals were considered adequate (Gerloff, 1992; Ellis et al., 1997) except for the control which was at a critical level (0.03 mg/L).

At 12 months after the start of the experiment, all selenium supplemented groups had higher ($P< 0.05$) plasma selenium concentrations than the control. The second free-choice supplement group was also higher ($P< 0.05$) than the other treatments. In the second summer (18 mo), the second free-choice supplementation group had higher ($P< 0.05$) plasma selenium than the other treatments, and all treatments were below suggested adequacy (0.07 mg/L), with the control and Mu-Se at a critical level (0.03 mg/L). At the end of the experiment (24 mo), there were also differences ($P<0.05$) among treatments with the free-choice mineral mixture treatments considered adequate

(0.07 mg/L), whereas the control treatment at 0.02 mg/L was below the critical level of 0.03 mg/L.

The control and the Mu-Se treatments had lower plasma selenium concentrations at the end of the study than at the start. The control started with 0.06 mg/L and ended with 0.02 mg/ L, suggesting that the selenium was being used by the animal at a greater rate than it was being replaced, but in this case no selenium was being supplemented. At the administered dose and frequency the Mu-Se source of selenium was not sufficient to raise and maintain adequate selenium concentrations in cow plasma. The Deposel treatment maintained the selenium status in cow plasma while both free-choice mineral mixture groups resulted in higher plasma selenium concentrations at the end of the study. These three treatments were more effective than the Mu-Se in raising plasma selenium concentrations in beef cows. It has been reported (Abdehlraman and Kincaid, 1995) that cows with daily intakes of 1 mg/d selenium were unable to maintain adequate selenium concentrations in blood during late gestation. Awadeh et al. (1997) found that cows consuming 1.27, 3.98 and 8.57 mg selenium per day had a decline in blood selenium post-partum, except for cows consuming the highest level.

In general, plasma selenium concentrations for the two mineral mixture treatments were adequate (0.07 mg/L) at the end of the experiment, not so for the Deposel and Mu-Se treatments which were below adequacy but above borderline (0.03 mg/L).

In a recent study, Ivancic and Weiss (2001) fed Holstein cows diets containing either 0.1 or 0.3 mg/kg of supplemental selenium from sodium selenate during 112 d. Plasma selenium concentrations were similar for both treatments (63 and 64 ug/L for 0.1 and 0.3 mg/kg selenium). The failure to elevate plasma selenium with inorganic sources has been reported previously (Maus et al., 1980). In the present study, something similar happened, when neither sodium selenite nor barium selenate were able to elevate plasma selenium concentrations. The best that happened was barium selenate maintaining the concentrations in plasma.

### Cow Liver Selenium

No differences ($P<0.05$) in liver selenium were found among treatments at the start of the experiment (Table 9). The cow liver selenium concentrations varied from 0.06 to 0.07 mg/kg, values which are below the 1.2 to 2.0 mg/kg considered to be normal liver selenium concentrations (Stowe and Herdt, 1992). The control was lower ($P<0.05$) than all other treatments. At six months, Mu-Se and Deposel treatment animals were higher ($P<0.05$) than the control but were lower ($P<0.05$) than for animals receiving the two free-choice mineral mixtures. Liver selenium concentrations for both groups of animals receiving the free-choice mineral mixtures were considered adequate for status of this element. Knowles et al. (1999) performed liver biopsies on cows that were provided selenium from sodium selenate or selenized yeast (Sel-Plex 50) after 133 d on the experiment. The selenium sources provided 2 or 4 mg/d selenium and the liver analyses revealed mean selenium concentrations of

Table 9. Liver selenium concentration (mg/kg) of Angus cows under different methods and sources of selenium supplementation (n = 75)[a]

| | Months of experiment | | | | |
|---|---|---|---|---|---|
| Sources | 0 | 6 | 12 | 18 | 24 |
| Control | $0.070^{b}$ | $0.041^{b}$ | $0.043^{b}$ | $0.034^{b}$ | $0.027^{b}$ |
| Sodium selenite (Mu-Se) | $0.073^{b}$ | $0.472^{c}$ | $0.612^{c}$ | $0.704^{c}$ | $0.923^{c}$ |
| Barium selenate (Deposel) | $0.070^{b}$ | $0.601^{c}$ | $0.826^{c}$ | $1.003^{d}$ | $1.694^{e}$ |
| Free-choice mineral # 1 (Se-yeast) | $0.075^{b}$ | $1.611^{e}$ | $1.358^{d}$ | $0.979^{d}$ | $1.287^{d}$ |
| Free-choice mineral # 2 (Se-yeast) | $0.063^{b}$ | $1.180^{d}$ | $1.304^{d}$ | $0.909^{cd}$ | $1.239^{d}$ |

[a] Data represent LS means and Standard error = 0.008.

[bcd] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

920, 1930, 2710, 2430, and 3920 nmol selenium/kg of liver fresh weight for control cows, cows treated with low or high concentrations of sodium selenate, and cows treated with low and high concentrations of selenized yeast, respectively. In our study, the organic selenium source (Se-yeast) was also able to increase selenium in liver, while the inorganic sodium selenite did not, which agrees with the above presented results. However, barium selenate in the present study was also able to raise selenium liver concentrations to adequate levels (1.2 - 2.0 mg/kg).

One year (12 mo) after the initiation of the experiment there were differences ($P<0.05$) among treatments. Liver selenium (0.04 mg/kg) in control animals was lower ($P<0.05$) than all other groups. The Mu-Se treatment (0.61 mg/kg) did not differ ($P<0.05$) from the Deposel treatment (0.83 mg/kg) and both treatments had less ($P<0.05$) liver selenium than the free-choice mineral groups (1.36 and 1.30 mg/kg), while these last two were not different among themselves. The control, Mu-Se and Deposel treatments were relatively low in selenium, whereas the two free-choice mineral mixes were adequate.

At 18 mo after initiation of experiment, there were differences ($P<0.05$) among treatments in liver selenium, with all treatments borderline to an adequate level of 1.2 mg/kg. The control was lower and different ($P<0.05$) than all the other treatments. The Mu-Se treatment differed ($P<0.05$) from the Deposel and free-choice mineral mix # 1 but was similar to free-choice mix # 2. The Deposel and free-choice mineral mix # 1 did not differ ($P<0.05$) among themselves.

At 24 mo (end of experiment), the Deposel treated animals had the highest liver selenium concentration (1.69 mg/kg) and were different ($P<0.05$) than all other groups followed by the two free-choice mineral mixture animals (1.29 and 1.24 mg/ kg). These three treatments had adequate selenium concentrations whereas the control and Mu-Se treatment did not.

The Deposel and two free-choice mineral mixture groups succeeded in raising liver selenium concentrations to adequate levels after two years (1.69, 1.29 and 1.24 mg/kg, respectively), and this is also reflected in plasma selenium concentrations of the cows, which were also adequate at the end of the experiment. The Mu-Se treatment did not reach adequate levels, but at two years had a tendency for higher selenium concentrations. These results have to be considered in the light that this treatment was administered every six months (recommendation is every 4 mo), whereas the Deposel was administered once at the start of the study, while the cattle on the free-choice treatment had continuous access to the selenium containing mineral.

The cows receiving barium selenate had the highest liver selenium at the end of the experiment. This accumulation of selenium in liver suggests that this slow release product could go on providing selenium to the animal for a longer period than two years, which was the duration of the experiment. However, if this is true, care should be taken at slaughter of culled older cows to avoid using the area where the Deposel was administered. The organic selenium (Se-yeast) also succeeded in providing adequate selenium levels at the end of the two year period, when mixed with the mineral salt. This is a practical way of supplementing selenium and one that is commonly used, as most cattlemen provide minerals to their cattle.

### Cow Colostrum Selenium

During the first year of the experiment there were differences ($P<0.05$) among treatments in colostrum selenium concentrations (Table 10) . The control treatment had the lowest value and was different ($P<0.05$) from all other treatments. Mu-Se was similar to Deposel but lower ($P<0.05$) than for animals receiving the free-choice mineral treatments. Abdehlraman and Kincaid (1995) have reported selenium concentrations in colostrum of 0.04 to 0.06 mg/L. The NRC (1996) has set values of 0.1 mg/kg as the dietary selenium requirements for young calves. Colostrum concentrations in this study are similar to those reported by Abdehlraman and Kincaid (1995) but below what is considered adequate for growing calves.

In the second year, colostrum selenium concentrations declined in all the treatments. The control presented the lowest selenium concentration (0.020 mg/L) but was not different ($P<0.05$) from the Mu-Se, Deposel and free-choice mineral mixture # 1. The highest selenium concentration was found in the free-choice mineral mixture # 2 (0.065 mg/L) and was different ($P<0.05$) from all other treatments except the Deposel treatment.

The tendency for the colostrum selenium concentrations in this trial is to decline from one year to the other. This would complicate the transfer of selenium from the dam to the calf because all of the concentrations are below the NRC (1996) standards.

Table 10. Colostrum selenium concentrations (mg/L) of Angus cows in two years under different sources and methods of selenium supplementation (n = 75)[a]

| | Years of experiment | |
|---|---|---|
| Sources | 1 | 2 |
| Control | 0.032[b] | 0.024[b] |
| Sodium selenite (Mu-Se) | 0.056[c] | 0.035[bc] |
| Barium selenate (Deposel) | 0.071[c] | 0.049[c] |
| Free-choice mineral #1 (Se-yeast) | 0.092[d] | 0.039[c] |
| Free-choice mineral # 2 (Se-yeast) | 0.092[d] | 0.065[d] |

[a] Data represent LS means and standard error = 0.007.

[bcd] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

The values found in the colostrum in this study are not enough to supply the calves with adequate selenium. It is noted that the average selenium in colostrum for the two free-choice mineral mixes at the end of the experiment (0.052 mg/L) was not very different ($P<0.05$) than the selenium in colostrum for the Deposel group (0.049 mg/L). According to Campbell et al. (1990), the selenium concentration in colostrum is significantly increased by supplementing, but the actual amounts of selenium are small and probably of little importance in the prevention of juvenile NMD.

### Cow milk Selenium

Ammerman et al. (1980) observed milk selenium concentrations in beef cows decreased from 0.015 mg/L two weeks after calving to 0.010 mg/L at eight weeks. Milk values ranging from 0.029 to 0.064 mg/L have been reported in dairy cows by Maus et al., (1980). At 60 days after calving the concentrations of selenium in the beef cows milk (Table 11) of the present study (0.02 to 0.04 mg/L) were above those reported by Ammerman et al. (1980) and similar to the lower range of values reported for dairy cattle (Maus et al., 1980). The highest concentrations were for the free-choice mineral mixture treatments (0.03 and 0.04 mg/L) and the lowest (0.02 mg/L) were found in the Mu-Se and Deposel groups.

At 120 days after treatment began, the tendency was similar, with the highest concentrations of selenium in milk being for the salt mineral mix treatments, with lower concentrations for the Deposel and Mu-Se treatments and the lowest for the control. At 180 days, again there was a similar trend. During the three periods, milk selenium

Table 11. Milk selenium concentrations (mg/L) of Angus cows under different methods and sources of selenium concentration (n = 75)[a]

| | 1997 | | | 1998 | | |
|---|---|---|---|---|---|---|
| | Days post-partum | | | Days post-partum | | |
| Sources | 60 | 120 | 180 | 60 | 120 | 180 |
| Control | $0.03^{cd}$ | $0.01^{b}$ | $0.01^{b}$ | $0.01^{b}$ | $0.01^{b}$ | $0.02^{c}$ |
| Sodium selenite (Mu-Se) | $0.02^{bc}$ | $0.02^{b}$ | $0.02^{bc}$ | $0.02^{c}$ | $0.02^{c}$ | $0.01^{b}$ |
| Barium selenate (Deposel) | $0.02^{b}$ | $0.02^{b}$ | $0.02^{bc}$ | $0.02^{c}$ | $0.01^{b}$ | $0.02^{c}$ |
| Free-choice mineral # 1 (Se-yeast) | $0.03^{de}$ | $0.04^{c}$ | $0.03^{c}$ | $0.02^{c}$ | $0.02^{c}$ | $0.02^{c}$ |
| Free-choice mineral # 2 (Se-yeast) | $0.04^{e}$ | $0.04^{c}$ | $0.03^{c}$ | $0.02^{c}$ | $0.02^{c}$ | $0.03^{d}$ |

[a] Data represent LS means and standard error = 0.002.

[bcd] Means with different superscripts within a column differ ($P<0.05$), by LSD test.

concentrations were maintained in all treatments, except for the control which decreased noticeably. All milk selenium concentrations were equal to but mostly above the values reported by Ammerman et al. (1980). These authors also reported a decline in milk selenium concentrations with time. Knowles et al. (1999) reported that selenized yeast was 2 to 3 times more effective than sodium selenite at increasing milk selenium concentration, and low intakes (2 mg/d selenium) of both supplements were 27 % more efficient per milligram of selenium administered than were high intakes (4 mg/d selenium). At day 133, milk selenium concentrations were 1.5 to 5.5 times higher in treated cows than in the control group. In the present study, during the first year selenized yeast was 2 times as effective as sodium selenite at 60 and 120 d in raising milk selenium concentrations and one time as effective at 180 d. During the second year of our study, all sources were equally effective but milk selenium concentrations were lower than in the first year.

During the second year, milk selenium concentrations at 60 days post-partum were low (0.02 mg/L) and presented no differences ($P<0.05$) among treatments but these values were higher than the control. At 120 days post-partum the control presented equal concentration of selenium (0.01 mg/L) as the Deposel treatment. The Mu-Se and both free-choice mineral mixture treatments were not different ($P<0.05$) but had a higher (0.02 mg/L) selenium concentration than the Deposel and control ($P<0.05$). At 180 days, milk selenium concentrations were highest (0.03 mg/L) for free-choice mineral mixture # 2 and lowest (0.01 mg/L) for the Mu-Se ($P<0.05$).

It has been demonstrated that placental transfer of selenium is effective in cattle because the maternal supplementation of cows in late gestation increases selenium reserves in the fetus as well as the newborn, as reported by Van Saun et al. (1989) and Abdelrahman and Kincaid (1995). Pre-partum maternal supplementation improves selenium status in calves to a greater extent than post-partum, suggesting that placental transfer of selenium is more efficient than milk transfer (Enjalbert, 1999). The selenium level in milk is readily raised by the supplementation of animal diets. Grant and Wilson (1968) obtained substantial milk selenium increases over a period of 3 to 4 weeks from cows receiving a single oral dose or subcutaneous dose of 50 mg selenium as selenate, while levels in untreated cows remained low. Gardner and Hogue (1967) tripled the selenium concentration of milk of ewes fed a low selenium diet when they supplemented the ewes diet with 2.25 mg per day selenium as selenite. In the present study, only during the first year were the selenium concentrations maintained in all treatments, except for the control which declined. In the second year of the study, only the Deposel and free-choice mineral treatment # 2 were able to maintain the selenium concentrations in milk. Milk selenium concentrations in this study were low and suggest that calves do not obtain adequate selenium through milk, which would indicate that it is not a good practice to supplement the dam post-partum for the purpose of increasing milk selenium. This is further supported by Campbell et al. (1990) who indicate that even though milk selenium is higher in supplemented groups, the actual amount of selenium in milk appears to be too little to be of any nutritional importance in a deficient neonate.

Ortman and Pehrson (1999) and Suoranta et al. (1993) have indicated that supplementing inorganic selenium to the dam to alleviate selenium needs in calves is not satisfactory due to the poor capacity of these compounds to increase the selenium content of milk. Ortman and Pehrson (1999) also report that organic selenium in the form of a yeast product for dairy cows results in higher concentrations of selenium in the milk than supplements of sodium selenite. In this study, however, even though the organic selenium was higher (($P<0.05$) than the sodium selenite treatment, the concentrations were not adequate (0.07 mg/L) although Bostedt and Schramel (1990) maintain that newborn calves need about 0.04 mg/L of selenium in plasma to meet their needs for normal growth and health. Perry et al. (1978) reported that selenium concentrations in milk were not a good indicator of dietary selenium.

## Summary

In a two year experiment, Angus cows were supplemented with selenium through salt free-choice mineral mixes (yeast form) and two injectable products, Deposel (barium selenate, long lasting) and Mu-Se (sodium selenite, short lasting). Plasma selenium levels increased in the two salt mineral mix treatments, whereas in the Mu-Se treatment the values decreased, while the Deposel was able to maintain selenium concentrations. In the control, plasma concentrations were below critical levels at the end of the experiment (24 mo) and at the critical level at 18 mo. In the Mu-Se treatment plasma values were at critical level at 18 mo and above critical but below adequate the rest of the time. The Deposel treatment attained adequacy (0.07 mg/kg) at 6 mo and at no time was at a critical level. Liver selenium concentrations of the control did not attain adequate levels during the 2 years, and even though the Deposel treatment reached adequacy only at 24 mo the trend for this treatment was to increase liver selenium concentrations with time. The Mu-Se treatment at no time presented adequate concentrations but tended to increase with time. The two free-choice salt mineral mixture treatments reached selenium adequacy in liver at 6, 12 and 24 mo after treatment began. Colostrum selenium concentrations decreased from the first to the second year, and at no time did any treatment reach adequacy (0.1 mg/L). Milk selenium concentrations declined from 60 to 180 days in the control during the first year, but the concentrations tended to be maintained in all treatments. During the second year, the selenium concentrations for the Mu-Se treatment declined, while the concentrations were maintained in the Deposel and both free-choice mineral mixture treatments. Milk selenium concentrations found in this study were not adequate (0.1 mg/L).

## Implications

The selenite (Mu-Se) treatment was not effective in raising plasma selenium concentrations while the barium selenate (Deposel) maintained a constant level of selenium in plasma. Neither the Mu-Se or Deposel treatments attained plasma selenium adequacy (0.07 mg/L) but were above the critical level (0.03 mg/L). The free-choice mineral mixture (Se-yeast) treatments did increase plasma selenium concentrations and attained adequacy. Liver selenium concentrations for the control and selenite (Mu-Se) treatment did not reach adequacy at any time, whereas the barium selenate (Deposel) treatment did so at 24 mo and the salt mineral mixes (Se-yeast) were adequate at 6, 12 and 24 mo. Colostrum selenium concentrations declined from the first to the second year. Concentrations in milk selenium were not found to be adequate and declined with time.

# CHAPTER 6
# SELENIUM STATUS OF BEEF CALVES FROM DAMS RECEIVING SELENIUM SUPPLEMENTATION

## Introduction

Suckling calves can be susceptible to selenium deficiency, especially when their dams receive insufficient dietary selenium. These calves often succumb to nutritional muscular degeneration (NMD), generally at birth or during the first few months of life, when they are rapidly growing. The use of inorganic selenium as a supplement to prevent deficiency has been suggested as being unsatisfactory due to the poor capacity of inorganic compounds to increase the selenium content of milk (Suoranta et al., 1993; Ortman and Pehrson, 1999).

A selenium deficiency can also produce premature births, weak or dead calves, as well as decreased immune response and "ill thrift" (Maas, 1983; Corah, 1991; McDowell, 1997). Newborn calves need about 0.04 ug/ml of selenium in plasma (Bostedt and Schramel, 1990) and > 2.2 ug of selenium/g of DM in liver (Van Saun et al. 1989) to meet their needs for normal growth and health. Close correlations exist between concentrations of selenium in blood of calves and in dams at birth (Kincaid and Hodgson, 1989), but little correlation is found between concentrations of selenium in the blood of dams and calves at 10 wks of age.

Dietary supplements of organic selenium for dairy cows, in the form of a yeast product, resulted in higher milk selenium than from supplements of inorganic selenium such as sodium selenite (Ortman and Pehrson, 1999). The aim of this study was to compare plasma selenium of growing calves for two years when their dams had been provided pre-partum selenium as a yeast product, sodium selenite (short-term effect) and barium selenate (long-term).

## Materials and Methods

Selenium status of Angus calves was evaluated over a 2-year period (1997 and 1998). In year 1 there were 54 calves, 29 males with an average birthweight of 35 kg and 25 females with an average birthweight of 33 kg. For year 2 there were 56 calves, 29 males with an average birthweight of 35 kg and 27 females with average birthweight of 34 kg. A 6 month trial was carried out each year at the Beef Demonstration Unit of the University of Florida at Chipley, Florida. The calves were born from February to April of each year and were from dams that had received one of the following treatments since December, 1996 : 1) a control (no selenium supplementation), 2) subcutaneous injection of 5 ml of Mu-Se (5 mg selenium per ml from sodium selenite, Burns Biotech Labs, Inc. Oakland, CA 94621) every 6 mo, 3) subcutaneous injection of 9 ml of Deposel (50 mg selenium per ml as barium selenate, Grampian Pharmaceuticals Ltd, Leyland, Lancashire PR53QN, U.K.) administered only at the initiation of the experiment, and, 4) use of a mineral mix with organic selenium (Se-yeast: Yea-Sacc, Alltech Biotech Center, 3031 Catnip Hill Pike, Nicholasville, KY

49356) administered to two groups as a replicate. The free-choice mineral mixture contained 30 mg/kg selenium, with cows on the two free-choice treatments averaging a consumption of 2.1 mg of selenium per day. All procedures of this study were approved by the Inter-institutional Animal Care and Use Committee (IACUC) of the University of Florida.

Blood from the calves was collected at birth and at 60, 120 and 180 days postpartum via jugular venipuncture with an 18 gauge needle into heparinized vacuum blood collection tubes. Plasma was separated by centrifugation and frozen for later analysis. Selenium plasma sample concentrations were determined using the Whetter and Ullrey (1978) fluorometric method.

The cows and their calves were provided bahiagrass (Paspalum notatum) and bermuda grass (Cynodon dactylon) pastures from May to October. During the winter (February to April) they grazed ryegrass (Lolium multiflorum) and were fed bahiagrass hay from November to February. Bahia and bermuda grass pastures analyzed 0.05 mg/kg selenium, ryegrass was 0.045 mg/kg and bahiagrass hay was 0.040 mg/kg (Chapter 5). The calves were weaned at 7-9 months.

The data were analyzed as a repeated measures model in a completely randomized design using the GLM of SAS (1966) and means compared by a LSD test.

## Results and Discussion

### Year One

Calf plasma selenium concentrations at birth in 1997 were different ($P<0.05$) among treatments (Table 12) with the free-choice mineral mixture # 2 treatment having adequate selenium plasma concentration (0.07-1.0 mg/L) and mixture # 1 not quite reaching adequacy, while the control and Deposel were at critical level (0.03 mg/L) and the Mu-Se was above borderline but not adequate. Pehrson et al. (1999) reported that Hereford calves whose dams received free access mineral supplement which contained 30 mg/kg selenium as Se-yeast or sodium selenite had plasma selenium concentrations of 0.047 and 0.036 mg/L, respectively. In the present study, where the free-choice mineral mixture contained 300 mg/kg Se-yeast the result was an average of 0.06 mg/L of plasma selenium concentration, which is borderline to adequate level. The calf plasma selenium concentrations of the inorganic treatments in this study are also similar to those of Pehrson et al. (1999). Toledo and Perry (1985) provided oral selenium supplementation to cows 60 days prepartum at 2 mg/head/d as sodium selenite and found that at birth calves from these cows had no different serum selenium concentrations than those whose dams had not received selenium (0.029 vs 0.025 mg/L, respectively). This agrees with the results of the present study where at birth, the control was not different ($P<0.05$) than the Mu-Se (sodium selenite) treatment (0.03 vs 0.04 mg/L). However, when McDowell et al. (1990) provided 0.6 % sodium selenite (0.25 ppm Se) as part of concentrate mix + i.m. injection of 5 mg selenium compared

Table 12. Calf plasma selenium concentrations (mg/L) at different ages whose Angus dams were supplemented with different sources of selenium during 1997 and 1998[a].

| | Intervals (d) | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | 1997 | | | | 1998 | | | |
| Sources | 0 | 60 | 120 | 180 | 0 | 60 | 120 | 180 |
| Control | $0.03^b$ | $0.02^b$ | $0.01^b$ | $0.02^b$ | $0.02^b$ | $0.02^b$ | $0.02^b$ | $0.02^b$ |
| Sodium selenite[1] (Mu-Se) | $0.04^b$ | $0.03^b$ | $0.02^{bc}$ | $0.02^b$ | $0.03^b$ | $0.03^b$ | $0.03^b$ | $0.02^b$ |
| Sodium selenate[2] (Deposel) | $0.03^b$ | $0.02^b$ | $0.03^b$ | $0.02^b$ | $0.03^b$ | $0.03^b$ | $0.02^b$ | $0.02^b$ |
| Free-choice mineral # 1 (Se-yeast)[3] | $0.05^c$ | $0.07^c$ | $0.09^d$ | $0.07^c$ | $0.05^c$ | $0.06^c$ | $0.07^c$ | $0.06^c$ |
| Free-choice mineral # 2 (Se-yeast)[3] | $0.07^d$ | $0.06^c$ | $0.08^d$ | $0.07^c$ | $0.06^c$ | $0.07^c$ | $0.06^c$ | $0.07^c$ |

[a] Data represent LS means and standard error = 0.004.

[bcd] Means with different superscripts within a column differ (P<0.05), by LSD test.

[1] Dams received a subcutaneous injection of 5 ml Mu-Se (Burns Biotech Labs, Inc.; 5 mg selenium per ml from sodium selenite) every 6 mo.

[2] Dams received a subcutaneous injection of 9 ml Deposel (Grampian Pharmaceuticals, Ltd.; 50 mg selenium per ml as barium selenate) administered once.

[3] Dams consumed a free-choice mineral mixture containing organic selenium (Se-yeast: Yea-Sacc, Alltech) which provided 54.5 g selenium per kg mixture.

to a mineral mixture with 0.25 mg/kg selenium in Brahman cows, they found that the two supplemental selenium treatments (0.053 vs 0.049 mg/L, respectively) were higher ($P<0.05$) than the control (0.030 mg/L), but not different ($P<0.05$) among themselves.

At 60 days postpartum the control, Mu-Se and Deposel treatments were not different ($P<0.05$) among themselves and were borderline or below the critical level of 0.03 mg/L (McDowell et al., 1984). The Mu-Se treatment was at a critical level while the control and Deposel at 0.02 mg/L were lower ($P<0.05$) than the two free-choice mineral mixture treatments. The two free-choice mineral mixture treatments were not different ($P<0.05$) among themselves with mixture # 1 being adequate in plasma selenium with 0.07 mg/L (Gerloff, 1992) and mixture # 2 borderline to adequate (0.06 mg/L). Abdelrahman and Kincaid (1995) found that at birth, calves of cows that had received selenium supplementation as sodium selenite had higher ($P<0.05$) plasma selenium concentrations (0.034 mg/L) than controls (0.028 mg/L), and at 42 d post-partum the values were still higher ($P<0.05$) for the calves from selenite treated cows (0.05 mg/L) compared to the control (0.04 mg/L). This is not in agreement with results of the present study where plasma selenium concentrations for the sodium selenite treatment tended to decrease at 60 days (0.04 to 0.03 mg/L), as did values reported by Pehrson et al. (1999), which decreased significantly ($P<0.02$) from 0.036 to 0.032 mg/L over 5 wk.

After 120 days, there were differences ($P<0.05$) among treatments. The two free-choice mineral mixture treatments were not different ($P<0.05$) among themselves (0.09

and 0.08 mg/L) and surpassed the adequate selenium level for plasma (0.07 mg/L) while being higher ($P<0.05$) than all other treatments. The Deposel treatment was at a critical level (0.03 mg/L) while the Mu-Se and control were below critical.

At 180 days postpartum, the inorganic treatments and the control were not different ($P<0.05$) and were below the critical level (0.03 mg/L). They were also lower ($P<0.05$) than the two free-choice mineral mixture treatments, and these two were similar ($P<0.05$) and attained selenium adequacy (0.07 mg/L).

**Year Two**

During 1998, the results show that the control was below critical level (0.03 mg/L). The Mu-Se and Deposel treatments were initially at the critical level (0 and 60 d) decreasing to below critical at 120 and 180 d. There was no difference ($P<0.05$) among the control and the Mu-Se and Deposel treatments during any time period. The two free-choice mineral mixtures, however, were higher ($P<0.05$) than all the other treatments but did not reach adequacy (0.07 mg/L) at any time. The free-choice mineral mixture # 1 presented selenium adequacy at 120 d with an average of 0.06 mg/L for the year while the free-choice mineral mixture # 2 was adequate at 60 and 180 d with an average of 0.065 mg/L for the year. There was no difference ($P<0.05$) between the two free-choice mineral mixtures.

In the present study, the trend is for the control group to have a decrease in plasma selenium which could be expected, because of no selenium supplementation. However, both inorganic treatments also tended to decrease in plasma selenium concentrations. Neither the Mu-Se (sodium selenite) nor the Deposel (slow release barium selenate) were able to increase selenium levels in plasma. This could possibly be attributed to the concentration of unsaturated fatty acids, mainly linolenic acid, in fresh grass that the dams were consuming from April to June. The cows consumption of fresh grass would increase, and so would their need of antioxidative substances to protect them against the oxidative effects of linolenic acid. The cows would thus draw on their selenium reserves and diminish their selenium at the expense of the calf. If this is true, then it becomes apparent that organic selenium is more effective in beef cattle to provide enough selenium for the dam and calf, as the calves plasma selenium concentrations in the two free-choice mineral mixture treatments were adequate during the duration of the experiment.

In beef cattle, selenium readily crosses the placenta and the fetus can usually attain selenium concentrations higher than those of the mother (Koller et al., 1984). Additionally, if dams are deficient in selenium, a higher concentration is observed in the calf. This could explain why control calves at birth didn't have a lower value than 0.03 mg/L reported. Fetal calves can partially compensate by concentrating selenium. This has also been found by Campbell et al. (1990). These authors also suggest that metabolism of the fetus may be minimal prior to birth resulting in a reserve. There may also exist a specific period during gestation in which selenium can be transferred across the placenta, and after that, it is trapped in the fetus. Abdelrahman and Kincaid (1995) reported that maternal supplementation at 3 mg/d of supplemental selenium as selenite via intraruminal bolus increased reserves in the liver of the newborn and in colostrum. Carryover effects of selenium supplementation were evident in calves at 42 d of age (Abdelrahman and Kincaid, 1995).

## Summary

Angus cows were supplemented with selenium (yeast- form) via salt free-choice mineral mixtures and two injectable products, Deposel (barium selenate, long term) and Mu-Se (sodium selenite, short term). The rates used are reported in Chapter 5. Plasma selenium of their calves was determined at birth and 60, 120 and 180 d postpartum. During year 1 (1997), calf plasma selenium concentrations at birth were at critical level (0.03 mg/L) but below adequacy for the control, Mu-Se and Deposel treatments. The two free-choice mineral treatments had an average concentration of 0.06 mg/L, which is borderline to adequate (0.07 mg/L). At 60, 120 and 180 d, the control, Mu-Se and Deposel treatments were below critical level concentrations, whereas the averages for the free-choice mineral mixtures were at or above adequacy. During year 2 (1998), the control was below critical level at all times, while Mu-Se and Deposel treatments were at critical level at birth and 60 d postpartum and below the critical level thereafter. The two free-choice mineral mixtures were higher ($P<0.05$) than all other treatments with average borderline concentrations (0.055 mg/L) at birth and adequate values (0.065 mg/L) the rest of the time. In general, the control and the two injectable inorganic treatments resulted in reduced calf plasma selenium concentrations with time and at no time were adequate. The two free-choice mineral mixture treatments with

organic selenium were adequate most of the time and, most important, were adequate at the end of the experiment (180 d).

## Implications

Neither injectable inorganic selenium source (Mu-Se or Deposel) was effective in raising and maintaining calf plasma selenium concentrations during this study and the trend was to decrease with time. The organic selenium treatments (two free-choice mineral mixtures) were effective in attaining and maintaining adequate selenium levels throughout the experiment. At the end of the study the calf plasma selenium concentrations were still adequate for the organic selenium treatments.

# CHAPTER 7
# SELENIUM CONCENTRATION OF FESCUE AND BAHIA GRASSES AFTER APPLYING A SELENIUM FERTILIZER

## Introduction

Most livestock regions in the USA, including the Southeastern states, are deficient in selenium, resulting in selenium responsive diseases when animals are not supplemented (Kubota et al., 1967). However, decreased productivity resulting from marginal selenium deficiency may be present in larger areas. Marginal selenium deficiency may be underestimated because clinical signs do not present themselves and a marginal deficiency may not happen every year or in all regions (Wilkins and Kilgour, 1982; Langlands et al., 1991).

Selenium provided as a fertilizer would be uniformly received by grazing livestock through forage consumption after topdressing pastures with selenium. This has proven to be effective in preventing selenium deficiency. This a common practice in livestock production in New Zealand (Watkinson, 1983) and in the high rainfall areas of Australia (Whelan, 1994) and is practically the only means of selenium supplementation in Finland (Gissel-Nielsen, 1993).

The selenate form of selenium, as compared with selenite, is the best to use in fertilization programs with both sodium and barium selenate utilized. Only a tenth as much selenate selenium is required, in contrast to selenite, to produce a given result

(Oldfield,1998). A selenium fertilizer, Selcote Ultra (Crop Care Holdings, Ltd., 25 McPherson St., Richmond 7002, Nelson, New Zealand), incorporating a slow release formula (90% minimum sodium and barium selenate and 10 % maximum sodium and barium selenite) is commercially available. Two experiments were conducted to determine the dynamics of selenium from this new fertilizer in fescue (Festuca spp.) and bahiagrass (Paspalum notatum) pastures at several rates of application by determining the selenium concentrations of grass tissue at different intervals of time.

## Materials and Methods

### Bahiagrass Experiment.

An experiment was conducted from August to November 1998 at the University of Florida in Gainesville (North Central Florida) to evaluate the selenium levels of bahia grass after spraying with a slow release selenium fertilizer, Selcote Ultra, a new slow-release selenium fertilizer incorporating 90 % minimum sodium and barium selenate and 10 % maximum sodium and barium selenite. Forage samples were collected from previously mowed 3 x 5 m plots (12 cm high) replicated 4 times which were sprayed with Selcote Ultra at rates of 0, 5, 10, 15, and 20 g/ha selenium. Samples were collected every 2 wk starting in August for a total of six collections. The soils had a pH of 5.7 and were classified as Millhopper sand. The selenium concentration of the bahiagrass before application ranged from 0.008 to 0.026 mg/kg. At each collection 1 $m^2$ of bahiagrass was clipped as near to the ground as possible and the samples were oven-dried at 55°C, ground through a 1 mm screen and stored in a freezer.

**Fescue Experiment.**

An experiment was conducted from January to July 1999 at the North Florida Research and Education Center of the University of Florida located at Quincy (Northwest Florida) to evaluate the selenium concentration of fescue grass after topdressing with Selcote Ultra. The fertilizer was applied at rates that would provide 0, 5, 10, 24, and 120 g/ha selenium (Valle et al., 1993). There were 4 replicates per treatment.

Soils in this area were classified as Orangeburg loamy fine sand (fine-loamy siliceous thermic Type Kandiudult), presenting a pH of 5.8. The monthly rainfall for the period from February to July was 57, 70, 16, 116, 145, and 192 mm, respectively.

The dry-granule fertilizer was broadcast by hand on 3 x 5 m plots which had been mowed. Forage samples were collected at 2, 4, 6, 10, 16, and 22 weeks after the application of the fertilizer. At each collection 1 $m^2$ of fescue was clipped as near to ground level as possible.

For both experiments samples were oven-dried at 55° C, ground through a 1 mm screen and stored in a freezer. Samples were analyzed for selenium according to the fluorometric procedure of Whetter and Ullrey (1978). The data obtained from four replicates were analyzed as a repeated measures experiment in a randomized complete block design using the GLM procedure of SAS (1996).

# Results

## Bahiagrass Experiment

Selenium concentrations of bahiagrass at different weeks after spraying with Selcote Ultra are presented in Table 13. Selenium concentrations (dry basis) were highest in the fourth and sixth wk for the 5,10 and 20 g/ha selenium treatments, respectively, while the 15 g/ha treatment presented the highest value at 2 wk after spraying, but declined thereafter. All treatments presented a marked decline in forage selenium concentrations at 8 wk after spraying and this trend continued at 12 wk.

The critical level of selenium suggested by the NRC (1996) for beef cattle is 0.1 mg/kg. Andrews et al. (1964) found that a concentration of 0.1 mg/kg is the minimum level needed to stop the occurrence of white muscle disease. Selenium concentrations in forage DM of the 15 g/ha treatment at 2 wk, of the 10, 15, and 20 g/ha treatments at 4 wk, and of the 15 and 20 g/ha treatments at 6 wk were the only ones meeting required levels for beef cattle. At 8 and 12 wk, none of the forage selenium concentrations were adequate.

## Fescue Experiment

Selenium concentrations in fescue DM at different wk after topdressing with Selcote Ultra can be observed in Table 14. Selenium was highest at 2 wk after applying Selcote Ultra, dropped off from wk 4 to 16 and at wk 22 the levels were still declining. The control (no added selenium) was below cattle requirements at all times. The four selenium treatments at all times presented adequate levels of forage selenium,

Table 13. Average selenium concentration (mg/kg, dry basis) of bahiagrass at different weeks after spraying with Selcote Ultra fertilizer[a].

| Levels of Se applied (g/ha) | Weeks after spraying | | | | |
|---|---|---|---|---|---|
| | 2 | 4 | 6 | 8 | 12 |
| 0 | $0.008^{b}$ | $0.026^{b}$ | $0.035^{b}$ | $0.009^{b}$ | $0.008^{b}$ |
| 5 | $0.019^{b}$ | $0.055^{bc}$ | $0.085^{bc}$ | $0.018^{b}$ | $0.008^{b}$ |
| 10 | $0.022^{b}$ | $0.246^{d}$ | $0.083^{bc}$ | $0.026^{b}$ | $0.015^{b}$ |
| 15 | $0.193^{c}$ | $0.168^{cd}$ | $0.173^{cd}$ | $0.071^{b}$ | $0.050^{b}$ |
| 20 | $0.080^{b}$ | $0.194^{d}$ | $0.235^{d}$ | $0.053^{b}$ | $0.025^{b}$ |

[a] Data represent LS means and standard error = 0.044.

[bcd] Means with different superscripts within a column differ (P<0.05), by LSD test.

Table 14. Average selenium concentration (mg/kg), dry basis) of fescue at different weeks after topdressing with Selcote Ultra fertilizer[a].

| Levels of Se applied (g/ha) | Weeks after applying | | | | | |
|---|---|---|---|---|---|---|
| | 2 | 4 | 6 | 10 | 16 | 22 |
| 0 | 0.06[b] | 0.06[b] | 0.07[b] | 0.05[b] | 0.05[b] | 0.03[b] |
| 5 | 1.90[bc] | 0.80[bc] | 0.32[b] | 0.26[b] | 0.25[b] | 0.14[b] |
| 10 | 2.42[bc] | 1.23[bc] | 0.91[b] | 0.27[b] | 0.28[b] | 0.17[b] |
| 24 | 4.04[c] | 3.14[cd] | 1.23[b] | 0.64[b] | 0.65[b] | 0.58[b] |
| 120 | 11.05[d] | 3.86[d] | 1.76[b] | 0.91[b] | 0.70[b] | 0.50[b] |

[a] Data represent LS means and standard error = 0.86.

[bcd] Means with different superscripts within a column differ (P<0.05), by LSD test.

except for the 24 and the 120 g/ha treatments which gave toxic levels of selenium, but only at 2 wk after fertilization.

## Discussion

The decline over time of bahiagrass selenium concentrations is in agreement with Archer (1983), who found that upon applying 170 g/ha of selenium as sodium selenate, grass DM samples contained up to 11.6 mg/kg selenium and clover up to 13.7 mg/kg in the first defoliation. This report demonstrates concentrations declining rapidly over time to a point that they were only slightly higher than those of untreated controls. On the other hand, when Rimmel et al. (1990) applied 10 and 20 g/ha of selenium to a grass-legume pasture, they found after collecting every 2 mo concentrations of 1.9, 1.1, 0.2, 0.19, 0.14, 0.11 for 10 g/ha and of 3.5, 2.1, 0.39, 0.41, 0.17 and 0.12 mg/kg selenium for 20 g/ha fertilizer selenium. These forage selenium levels were adequate without reaching toxic levels and the samples came from 6 harvests over 12 months. In the above study, forage DM had adequate (0.1 mg/kg) selenium concentrations one year after application.

Ekholm et al. (1991) studied the effects of different types of selenium supplementation on muscle and internal organs of bovines. They kept 32 bulls in 4 dietary groups from birth until 13-14 mo, and fed rations containing either 0.03 mg selenium/kg (basic level in local feeds), 0.25 mg inorganic selenium supplemented as sodium selenite, or 0.25 or 0.40 mg/kg plant selenium in dry matter from forage produced by spraying with sodium selenite. Plant selenium at both concentrations

(0.25 and 0.40 mg/kg) was more effective then sodium selenite in raising the selenium content of muscle tissues. Muscle tissue and liver were high responders to plant selenium. Liver increased 7.4 fold (from 0.23 mg/kg in the control to 1.7 mg/kg in the 0.40 mg/kg plant selenium group) while top round muscle increased 12 fold from 0.08 mg/kg to 0.96 mg/kg, respectively.

The selenate in fertilizer is presumably mainly transformed to organic compounds in plants (Gissel-Nielsen and Bisbjerg, 1970). Cereals and forage crops convert selenium mainly into Se-Met and incorporate it into protein in place of methionine (Met) because tRNA$^{Met}$ does not discriminate between Met and Se-Met (Schrauzer, 2000). Se-Met is not required for growth by plants but is produced along with Met in quantities depending on the amount of selenium available. In plant tissue of the grassland legume species *Melilotus indica L.*, for example (Guo and Wu, 1998), Se-Met contents increased with increasing soil concentrations of selenium until Se-Met accounted for >50 % of the total selenium content of the plant. Because higher animals have no efficient mechanism for Met synthesis, they are also unable to synthesize Se-Met (Schrauzer, 2000). Thus, plants fertilized with selenium are an important source of Se-Met for cattle. The risk of toxicity is minimized because at constant intakes in the nutritional range, tissue selenium levels increase until a steady state is established, preventing the buildup to toxic levels (Schrauzer, 2000). It is easier to prevent selenium deficiencies in livestock on deficient soils by supplying selenium by different methods than to prevent toxicity on seleniferous soils. Elemental selenium is preferred in heavy boluses, sodium selenite is preferred in other oral or parenteral applications, and sodium selenate or barium selenates are used as fertilizer amendments (Oldfield, et al.1994). Soil amendment by addition of selenium fertilizers has been shown to be an effective means of avoiding selenium deficiency in diets of animals and humans. Two countries, Finland (Ekholm, 1997; Eurola et al., 1989) and New Zealand (Gupta and Watkinson, 1985; Shirer, 1998) which have major land areas considered selenium-deficient, have gained extensive experience in this technology. An application of 10 g/ha selenium can raise forage crops level of selenium sufficiently to meet the needs of livestock, as reported by Valle et al. (1993) and reviewed by Gupta and McLeod (1998).

Valle et al. (1993) reported that selenium concentrations in bermudagrass DM reached non-toxic levels between 6 to 12 wk after spraying with sodium selenate, and that 24 g/ha selenium is an application rate that can be used safely to provide selenium to grazing livestock. For that study, sodium selenate was favored as the supplemental form instead of selenite because only 1/5 as much is required to raise pasture to the same selenium concentration (Grant, 1965), and it is safer since foliar contamination effects are therefore also about five times less (Grant, 1969; Thacker, 1961).

Selenium fertilization should ideally maintain forage levels between 0.1 and 4 mg/kg because selenium deficiency is reported when feeds contain less than 0.1 mg/kg (Andrews et al., 1964; Allaway et al., 1967), and toxicity if diets are above 5 mg/kg (Thacker, 1961). However, the maximum tolerable in cattle is controversial, to say the least. Ellis et al. (1997) report that Holstein cows consuming inorganic selenium (sodium selenite) at intakes of up to 50 mg/d for 90 d or 100 mg/d for 28 d tolerated these levels without adverse clinical consequences. At 2 wk after spraying, in the present bahiagrass experiment, only the 15 g/ha treatment provided adequate forage selenium levels, whereas at 4 and 6 wks the 10, 15 and 20 g/ha treatments allowed adequate forage selenium concentrations. All treatments declined to inadequate forage levels at 8 and 12 weeks after spraying.

The variable results encountered between this study and others can be attributed to several reasons. It has been reported that pasture selenium concentrations shortly after fertilization seem to be a property more of the plant and the chemical form of the selenium applied than of soil characteristics (Grant, 1965; Davies and Watkinson, 1966), with grasses absorbing more from the roots and legumes more from the leaves (Watkinson and Grant, 1981). In this study the selenium was sprayed upon the grass and it could be that bahiagrass is not able to absorb much selenium through the foliage, in agreement with Watkinson and Grant (1981). This is supported by the fact that both the matrix and barium selenate of the Selcote Ultra are largely insoluble in water, and are only released in the soil by the action of soil acids and microbes. The use of this product in a spray would be very inefficient as only the soluble portion of the selenium would be released, and 75 % of the selenium would remain in the gritty, insoluble portion of the material (Shirer, 2000).

The results of the fescue experiment are in agreement with Rimmel et al. (1990) who applied 10 and 20 g/ha selenium to a grass-legume pasture and reported DM concentrations of 1.9 to 0.11 mg/kg for the 10 g treatment and from 3.5 to 0.12 mg for the 20 g treatment over a one year period. Based on the levels obtained in the present study for fescue, it could be possible that after one year the forage selenium would still be adequate. The levels of forage selenium for 10 and 24 g/ha selenium are higher at 10 wk and at 22 wk than the ones reported by Rimmel et al. (1990) at 2 and 4 mo.

The use of Selcote Ultra as a means of providing selenium to forages was investigated by Whelan (1994), who found that the slow release Selcote continued to provide adequate selenium to the pasture in the second and third years of their study after an initial application of 10 and 20 g/ha selenium. For this study, the higher rate of application provided the highest concentration of forage selenium. Archer (1983) reports that after applying 170 g/ha selenium as sodium selenate, grass DM contained up to 11.6 mg/kg selenium and clover up to 13.7 mg/kg in the first defoliation. These values are considered toxic, but by the end of the first growing season they were only slightly higher than those of untreated controls.

Shirer (2000) indicates that, with regard to toxicity, its necessary to distinguish between chronic and acute toxicity. The higher levels obtained in the present study would probably only be toxic if animals consumed grass year round. Grazing animals would only be exposed to higher levels for a period of time, and it should be noted that those rates above 20 g/ha of selenium would be too expensive for practical purposes.

Valle et al. (1993) sprayed selenium as sodium selenate on bermudagrass at rates of 0, 24, 120, 240, and 480 g/ha and collected grass samples at 2, 4, 6, 12, and 18 wk after spraying. The selenium concentrations for the fertilized level of 24 g/ha selenium at no time resulted in any toxic concentrations for livestock. However, application rates of 120, 240, and 480 g/ha caused toxic levels in forage DM for more than 6 wk after spraying, but by wk 12 concentrations were down to a harmless and adequate concentration of 0.48-0.75 mg/kg.

Provision of selenium through pasture fertilizer has many advantages. It avoids labor costs of handling animals for individual treatments, its easier to treat larger animals such as cattle and it is available to both ruminant and monogastric animals (Whelan, 1994). Management of a pasture, however, can affect selenium available to animals grazing it. Aeration and increased soil fertility, as when fertilized, can decrease levels of selenium through mineral-diluting effect of rapid pasture growth (Wichtel, 1998). In winter, selenium is released from soil and is available for crops in the spring, when the yield of grass is low, and this results in a relatively high concentration of selenium. As the growth of grass increases during summer, however, less selenium is available for plants, and it is distributed into a much bigger yield. Thus, the concentration in grass drops as a result of dilution (Gissel-Nielsen, 1993). This could explain the decline in time of the selenium concentration in the fescue of this study. At the start, in January and February, selenium concentrations were high but started to decline towards the summer. At no time, however, were these concentrations deficient or inadequate for the selenium treatments.

Bahiagrass and fescue did not respond similarly to selenium fertilization. According to Grant (1965) and Davies and Watkinson (1966), pasture selenium concentrations after fertilization are more a property of the plant and the chemical form of selenium that has been applied than of soil characteristics. Elsewhere, Higgins and Fey (1993) found selenium deficiencies in ryegrass pastures in Natal and no soil properties could be identified by multiple regression as causing such a deficiency. In this study, the response was different in concentrations attained and in the time that these levels were adequate for beef cattle (0.1 mg/kg). Bahiagrass provided adequate levels of forage selenium only at 4 and 6 wk after fertilization with 10, 15, and 20 g/ha selenium. At 8 and 12 wk after fertilization the selenium concentrations of the forage were not adequate to meet beef cattle requirements. On the other hand, fescue selenium concentrations were always adequate for all treatments and at 2 wk after fertilization the 24 and 120 g/ha selenium treatments had toxic levels of selenium in forage. After 2 wk, no toxic levels of selenium were found in any treatment.

Several authors have reported similar differences among grass species (Davies and Watkinson, 1966; Grace and Clark, 1991; Mayland, 1994). In a comparison between kikuyo and ryegrass, Van Ryssen et al. (1998) found that ryegrass seems to have a much better ability than kikuyo to take up selenium from the soil. Additionally, selenium concentration in unfertilized ryegrass was higher than that of kikuyo. It could be that bahiagrass is similar to kikuyo in its ability to take up selenium from the soil and fescue more similar to ryegrass. Also, seleniuim fertilization did not increase selenium level in kikuyo significantly above the level in unfertilized kikuyo, compared to ryegrass which showed a dramatic increase ($P<0.01$) in selenium after fertilization, as reported by Van Ryssen et al. (1998). In the bahiagrass study selenium was sprayed upon the grass and it could be that the plant is not able to absorb much selenium through the foliage, in agreement with Watkinson and Grant (1981), whereas in the fescue study the selenium fertilizer was topdressed and was readily absorbed by the plant from the soil. One of the factors influencing selenium uptake is the pH of the soil, with selenium being more available to the plant at a high than a low pH (Higgins and Fey, 1993).

## Summary

Two experiments were conducted, one with bahiagrass in Northcentral Florida (Gainesville), and the other with fescue in Northwest Florida (Quincy) to evaluate the selenium levels of the grasses after applying a slow release selenium fertilizer (Selcote Ultra). In the bahiagrass study forage samples were collected from 3 x 5 m plots which had been sprayed with Selcote Ultra at treatment rates of 0, 5, 10, 15 and 20 g/ha selenium. Samples were collected every two weeks for a total of six collections. In the fescue study the procedure was the same except that forage samples were collected at 2, 4, 6, 10, 16, and 22 wk after applying the fertilizer, and the rates were 0, 5, 10, 24, and 120 g/ha selenium. For bahiagrass, only the 10, 15 and 20 g/ha selenium treatments

reached acceptable levels in the forage (>0.1 mg/kg) for beef cattle. The 10 g/ha selenium treatment reached adequate levels (0.25 mg/kg) only at 4 wk after application. The 15 g/ha selenium treatment presented adequate levels of selenium at 2, 4 and 6 wk after application (0.19, 0.17 and 0 .17 mg/kg, respectively). The 20 g/ha selenium treatment had adequate forage selenium at 4 and 6 wk after application ( 0.19 and 0.23 mg/kg, respectively). The 5 g/ha selenium treatment and the control did not reach acceptable values and were selenium deficient. All treatments were deficient at 8 and 12 wk. In the fescue study, all treatments presented adequate concentrations (0.1 mg/kg) of selenium in the forage, except for the control. At two weeks after application, the 24 g/ha and the 120 g/ha selenium treatments had toxic selenium levels in forage. However, these values declined to below toxicity (4-5 mg/kg) at all other collection dates.

## Implications

Spraying of Selcote Ultra fertilizer on bahiagrass during the summer is not very effective in raising forage selenium to adequate levels (> 0.1 mg/kg) beyond 8 wks for the 5 g/ha rate while the 10, 15 and 20 g/ha rate treatments were still adequate at 8 wks. However, topdressing of Selcote Ultra on fescue is an adequate method to increase forage selenium as the selenium concentrations were more than adequate 22 wk after the application.

# CHAPTER 8
# SUMMARY AND CONCLUSIONS

Several studies were conducted in north Florida to evaluate methods and sources of selenium supplementation in beef cattle. A two-year study was implemented in Chipley, Florida to evaluate two injectable selenium sources, Mu-Se (sodium selenite) and Deposel (barium selenate) as well as an organic source (Se-yeast) by determining tissue (liver, blood, colostrum and milk) selenium concentration of Angus cows. Also, Angus steers and heifers were used during an 84 d experiment to determine their weight gains as well as liver and blood selenium concentrations when fed two sources (inorganic and organic) of selenium at two levels. At the end of the steer and heifer experiment, half of the animals were fed low selenium containing hay and then subjected to a thyrothropin-releasing-hormone (TRH) challenge and their blood analyzed for $T_3$ and $T_4$ levels to determine the effect of the selenium sources on thyroid hormone metabolism. Additionally, two grasses, bahia-grass and fescue, were sprayed and topdressed with Selcote Ultra, a new slow release selenium fertilizer in two separate experiments to determine the dynamics of selenium in these grasses and evaluate the potential of these practices as methods of providing selenium to grazing cattle.

When Angus steers and heifers where supplemented with sodium selenite or Se-yeast at two levels (0.1 and 0.3 mg/kg in the feed), the organic selenium source proved to be more effective in stimulating weight gains and increasing liver selenium concentrations than the inorganic source. Organic selenium at 0.1 mg/kg stimulated the highest weight gains while organic selenium at 0.3 mg/kg resulted in the highest liver selenium concentrations. There were differences ($P<0.01$) in serum selenium concentrations attributed to selenium source, with the organic selenium resulting in higher concentrations, although none of the treatments reached adequate levels in the serum.

Supplementation with sodium selenite or Se-yeast did not alter the plasma selenium concentrations of $T_3$ and $T_4$ in Angus steers and heifers and the inorganic or organic selenium sources had no apparent effect on thyroid hormone metabolism.

Seventy-five Angus cows (511 kg average weight, 5-8 mo pregnant) were utilized in a two year experiment at the Beef Demonstration Unit of the University of Florida at Chipley, Florida. Treatments consisted of the following: 1) a control (no selenium supplementation), 2) sub-cutaneous injection of 5 ml of Mu-Se (5 mg selenium per ml from sodium selenite) every six mo, 3) sub-cutaneous injection of 9 ml of Deposel (50 mg selenium per ml as barium selenate) administered only at the initiation of the experiment, and 4) use of a free-choice mineral mix with organic selenium (Se-yeast: Yea-Sacc, Alltech) administered to two groups (replicates0 of cows. The free-choice mineral mixture contained 300 mg/kg selenium. The selenite

(Mu-Se) treatment was not effective in raising plasma selenium concentrations while the barium selenate (Deposel) maintained a constant level of selenium in plasma. Neither the Mu-Se or Deposel treatments attained suggested plasma selenium adequacy (0.07 mg/L) but were above the critical level (0.03 mg/L). The free-choice mineral mixture (Se-yeast) treatments did increase plasma selenium concentrations and attained adequacy. Liver selenium concentrations for the control and selenite (Mu-Se) treatment did not reach adequacy at any time, whereas the barium selenate (Deposel) treatment did so at 24 mo and the salt mineral mixtures (Se-yeast) were adequate at 6, 12 and 24 mo. Colostrum selenium concentrations declined from the first to the second year. Milk selenium concentrations were not found to be adequate and declined with time.

Plasma selenium concentrations of calves born to dams in the above two-year study were determined to evaluate effect of the selenium treatments on calf selenium adequacy. Fifty four Angus calves, of which twenty-nine were males with average birth weights of 35 kg and twenty-five were females with average birth weights of 33 kg, were utilized during the first year. In the second year, fifty-six calves were used, twenty-nine males with an average birth weight of 35 kg and twenty-seven females with average birth weights of 34 kg. Blood from the calves was collected at birth and at 60, 120, and 180 d post-partum. Neither injectable inorganic selenium source (Mu-Se or Deposel) given to the dams was effective in raising and maintaining calf plasma selenium concentrations during this study and the trend was to decrease with time. The organic selenium treatments (two free-choice mineral mixtures) were effective in attaining and maintaining adequate selenium levels throughout the experiment. At the end of the study the calf plasma selenium concentrations were still adequate for the organic selenium treatments. Therefore, organic selenium supplements would be a good choice to supply selenium for cattle in selenium deficient soils.

Two forage experiments were conducted, one with bahiagrass in Northcentral Florida (Gainesville) and the other with fescue in Northwest Florida (Quincy), to evaluate the selenium levels of the grasses after applying a slow release selenium fertilizer (Selcote-Ultra). The bahia grass was sprayed with Selcote-Ultra at rates of 0, 5, 10, 15 and 20 g/ha selenium and samples were collected every two weeks, for a total of six collections. In the fescue study, the forage samples were collected at 2, 4, 6, 10, 16 and 22 wks after topdressing the fertilizer Selcote-Ultra, and the rates were 0, 5, 10, 24 and 120 g/ha selenium. Spraying of Selcote-Ultra on bahiagrass was not very effective in raising forage selenium to adequate levels (>0.1 mg/kg) beyond 8 wks for the 5 g/ha rate while the 10, 15 and 20 g/ha rate treatments were still adequate at 12 wks. Topdressing of Selcote-Ultra on fescue was an effective method to increase forage selenium as the selenium concentrations were more than adequate 22 wks after the application.

# APPENDIX

Table A-1. ANOVA of average daily gains (kg) of Angus calves supplemented with organic and inorganic selenium (n = 40).

| Source of variance | df | Sum of squares | Mean square | F value | Required F 0.05 | Required F 0.01 |
|---|---|---|---|---|---|---|
| Treatment | 4 | 3.74 | 0.94 | | | |
| Source | 1 | 0.96 | 0.96 | 2.83NS | 4.12 | 7.41 |
| Level | 1 | 1.49 | 1.49 | 4.41* | 4.12 | 7.41 |
| Source x level | 1 | 0.002 | 0.002 | 0.01NS | 4.12 | 7.41 |
| Control vs others | 1 | 1.29 | 1.29 | 3.83NS | 4.12 | 7.41 |
| Error (a) | 35 | 11.83 | 0.34 | | | |
| Time | 2 | 36.08 | 18.04 | | | |
| Time x treatment | 8 | 5.96 | 0.74 | 1.55NS | 2.07 | 2.77 |
| Time x source | 2 | 2.61 | 1.30 | 2.72NS | 3.13 | 4.92 |
| Time x level | 2 | 0.61 | 0.30 | 0.63NS | 3.13 | 4.92 |
| Time x source x level | 2 | 0.16 | 0.08 | 0.17NS | 3.13 | 4.92 |
| Control vs others | 2 | 2.58 | 1.29 | 2.69NS | 3.13 | 4.92 |
| Error (b) | 70 | 33.55 | 0.48 | | | |

NS Non significant
* Significant ($P<0.05$)

Table A-2. ANOVA of plasma selenium concentrations (ug/ml) of Angus calves supplemented with organic or inorganic selenium (n = 40).

| Source of variance | df | Sum of squares | Mean square | F value | Required F<br>0.05 | <br>0.01 |
|---|---|---|---|---|---|---|
| Treatment | 4 | 0.005 | 0.001 | | | |
| Source | 1 | 0.002 | 0.0023 | 29.03** | 4.12 | 7.41 |
| Level | 1 | 0.0008 | 0.0008 | 9.89** | 4.12 | 7.41 |
| Source x level | 1 | 0.00000012 | 0.00000012 | 1.00NS | 4.12 | 7.41 |
| Control vs others | 1 | 0.00167703 | 0.00167703 | 21.53NS | 4.12 | 7.41 |
| Error (a) | 35 | 0.0027 | 0.00007789 | | | |
| Time | 3 | 0.00433462 | 0.00144487 | 13.90** | 2.70 | 3.98 |
| Time x treatment | 12 | 0.00772279 | 0.00064357 | 6.19** | 1.85 | 2.36 |
| Time x source | 3 | 0.00167153 | 0.00055718 | 5.36** | 2.70 | 3.98 |
| Time x level | 3 | 0.00110941 | 0.00036980 | 3.56* | 2.70 | 3.98 |
| Time x source x level | 3 | 0.00136569 | 0.00045523 | 4.38** | 2.70 | 3.98 |
| Control vs others | 3 | 0.00357616 | 0.00119205 | 11.46** | 2.70 | 3.98 |
| Error (b) | 105 | 0.10917841 | 0.00010398 | | | |

NS Non significant.
* Significant (P<0.05).
** Highly significant (P<0.01).

Table A-3. ANOVA of liver selenium concentrations (ug/g) of Angus calves supplemented with organic and inorganic selenium (n = 40).

| | | | | | Required F | |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean square | F value | 0.05 | 0.01 |
| Treatment | 4 | 0.7184 | 0.1796 | 7.27** | 2.64 | 3.91 |
| Source | 1 | 0.0930 | 0.0930 | 5.71* | 4.12 | 7.41 |
| Level | 1 | 0.5148 | 0.5148 | 31.58** | 4.12 | 7.41 |
| Source x level | 1 | 0.0410 | 0.0410 | 2.52NS | 4.12 | 7.41 |
| Control vs others | 1 | 0.0696 | 0.0696 | 4.27* | 4.12 | 7.41 |
| Error (a) | 35 | 0.5706 | 0.5706 | | | |
| Time | 1 | 0.1256 | 0.1256 | 5.09* | 4.12 | 7.41 |
| Time x treatment | 4 | 0.8109 | 0.2027 | 8.21** | 4.12 | 7.41 |
| Time x source | 1 | 0.2425 | 0.2425 | 9.82** | 4.12 | 7.41 |
| Time x level | 1 | 0.0961 | 0.0961 | 3.89NS | 4.12 | 7.41 |
| Time x source x level | 1 | 0.0056 | 0.0056 | 0.23NS | 4.12 | 7.41 |
| Control vs others | 1 | 0.4666 | 0.4666 | 18.90** | 4.12 | 7.41 |
| Error (b) | 35 | 0.8643 | 0.0247 | | | |

NS Non significant.
* Significant ($P<0.05$).
** Highly significant ($P<0.01$).

Table A-4. ANOVA of $T_3$ plasma selenium concentrations (ng/ml) of Angus calves supplemented with organic and inorganic selenium (n = 20).

| Sources of variance | df | Sum of squares | Mean square | F value | Required F 0.05 | Required F 0.01 |
|---|---|---|---|---|---|---|
| Treatment | 4 | | | | | |
| Source | 1 | 0.0006 | 0.0006 | 0.008NS | 18.00 | 90.00 |
| Levels | 1 | 0.209 | 0.209 | 2.863NS | 18.00 | 90.00 |
| Source x level | 1 | 0.002 | 0.002 | 0.027NS | 18.00 | 90.00 |
| Control vs others | 1 | 0.004 | 0.004 | 0.055NS | 18.00 | 90.00 |
| Error (a) | 5 | 0.365 | 0.073 | | | |
| Time | 7 | 1.064 | 0.152 | 5.850** | 3.28 | 5.75 |
| Time x levels | 28 | 0.682 | 0.024 | 0.920NS | 1.72 | 2.18 |
| Error (b) | 35 | 0.910 | 0.026 | | | |

NS Non significant.
** Highly significant (P<0.01).

Table A-5. ANOVA of $T_4$ plasma concentrations (ng/ml) of Angus calves supplemented with organic and inorganic selenium (n = 20).

| | | | | | Required F | |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean square | F value | 0.05 | 0.01 |
| Treatment | 4 | 789.77 | 197.44 | 1.510NS | 5.91 | 14.37 |
| Source | 1 | 24.13 | 24.13 | 0.185NS | 5.91 | 14.37 |
| Levels | 1 | 5.46 | 5.46 | 0.042NS | 5.91 | 14.37 |
| Source x level | 1 | 197.05 | 197.05 | 1.511NS | 5.91 | 14.37 |
| Error (a) | 5 | 652.19 | 130.44 | | | |
| Time | 7 | 1263.99 | 180.57 | 8.360** | 3.28 | 5.75 |
| Times x levels | 28 | 878.55 | 31.38 | 1.450NS | 1.72 | 2.18 |
| Error (b) | 35 | 755.72 | 21.59 | | | |

NS Non significant.

** Highly significant (P<0.01).

Table A-6. ANOVA of plasma selenium concentrations (mg/L) of Angus cows supplemented with organic and inorganic selenium (n = 75).

| | | | | | Required F | |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean square | F value | 0.05 | 0.01 |
| Treatment | 4 | 0.062 | 0.016 | | | |
| Source | 1 | 0.031 | 0.031 | 103.33** | 2.50 | 3.55 |
| Levels | 1 | 0.005 | 0.005 | 16.67** | 2.50 | 3.55 |
| Error (a) | 70 | 0.020 | 0.0003 | | | |
| Time | 4 | 0.025 | 0.006 | 30.00** | 3.02 | 3.90 |
| Time x level | 16 | 0.043 | 0.003 | 15.00** | 3.43 | 4.34 |
| Times x source | 3 | 0.013 | 0.004 | 20.00** | 2.92 | 3.80 |
| Times x level | 3 | 0.014 | 0.005 | 25.00** | 2.92 | 3.80 |
| Times x source x level | 3 | 0.003 | 0.001 | 5.00** | 2.92 | 3.80 |
| Control vs others | 3 | 0.007 | 0.002 | 10.00** | 2.92 | 3.80 |
| Error (b) | 280 | 0.058 | 0.0002 | | | |

** Highly significant (P<0.01).

Table A-7. ANOVA of liver selenium concentrations (ug/g) of Angus cows supplemented with organic and organic selenium (n = 75).

| Sources of variance | df | Sum of squares | Mean squares | F value | Required F 0.05 | Required F 0.01 |
|---|---|---|---|---|---|---|
| Treatment | 4 | 33.25 | 8.31 | 90.32** | 3.10 | 4.10 |
| Source | 1 | 0.40 | 0.40 | 4.35** | 2.61 | 3.83 |
| Levels | 1 | 4.28 | 4.28 | 82.30** | 2.61 | 3.83 |
| Source x level | 1 | 2.26 | 2.26 | 24.56** | 2.61 | 3.83 |
| Control vs others | 1 | 26.31 | 26.31 | 285.98** | 2.61 | 3.83 |
| Error (a) | 45 | 4.17 | 0.092 | | | |
| Times | 4 | 26.76 | 6.69 | 83.00** | 3.02 | 3.90 |
| Times x levels | 16 | 15.61 | 0.97 | 12.60** | 1.67 | 2.04 |
| Times x source | 3 | 0.28 | 0.09 | 1.95NS | 2.92 | 3.80 |
| Times x levels | 3 | 4.67 | 1.56 | 33.91** | 2.92 | 3.80 |
| Times x levels x sources | 3 | 0.54 | 0.18 | 3.91 | 2.92 | 3.80 |
| Control x others | 3 | 5.26 | 1.75 | 38.04** | 2.92 | 3.80 |
| Error (b) | 180 | 8.28 | 0.046 | | | |

NS Non significant.

** Highly significant (P<0.01).

Table A-8. ANOVA of colostrum selenium concentrations (mg/L) of Angus cows supplemented with organic and inorganic selenium (n = 75).

| | | | | | Required F | |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean square | F value | 0.05 | 0.01 |
| Treatment | 4 | | | | | |
| Source | 1 | 0.003 | 0.003 | 10.50** | 2.87 | 3.85 |
| Level | 1 | 0.005 | 0.005 | 16.00** | 2.87 | 3.85 |
| Source x level | 1 | 0.0000001 | 0.0000001 | 0.02NS | 2.87 | 3.85 |
| Control vs others | 1 | 0.015 | 0.015 | 50.00** | 2.87 | 3.85 |
| Error (a) | 35 | 0.010 | 0.0003 | | | |
| Times | 1 | 0.014 | 0.014 | 31.11** | 2.87 | 3.85 |
| Times x levels | 4 | 0.005 | 0.0012 | 2.66NS | 3.11 | 4.12 |
| Error (b) | 35 | 0.016 | 0.00045 | | | |

NS Non significant.
** Highly significant (P<0.01).

Table A-9. ANOVA of milk selenium concentrations (mg/L) in Angus cows supplemented with organic and inorganic selenium (n = 75).

| | | | | | Required F | |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean square | F value | 0.05 | 0.01 |
| Treatment | 4 | | | | | |
| Source | 1 | 0.00002 | 0.00002 | 0.21NS | 2.87 | 3.85 |
| Level | 1 | 0.004 | 0.004 | 43.01** | 2.87 | 3.85 |
| Source x level | 1 | 0.0002 | 0.0002 | 2.15NS | 2.87 | 3.85 |
| Control vs others | 1 | 0.0016 | 0.0016 | 17.20** | 2.87 | 3.85 |
| Error (a) | 35 | 0.0032 | 0.000093 | | | |
| Times | 5 | 0.0057 | 0.0011 | 12.22** | 3.09 | 3.98 |
| Times x levels | 20 | 0.0054 | 0.0003 | 3.33NS | 3.47 | 4.41 |
| Error (b) | 175 | 0.0136 | 0.000077 | | | |

NS Non significant.
** Highly significant (P<0.01).

Table A-10. ANOVA of plasma selenium concentrations (mg/L) of Angus calves whose dams where supplemented with organic and inorganic selenium (n = 60).

|  |  |  |  |  | Required F |  |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean squares | F value | 0.05 | 0.01 |
| Treatments | 4 |  |  |  |  |  |
| Source | 1 | 0.000002 | 0.000002 | 0.022NS | 2.92 | 3.96 |
| Level | 1 | 0.0667 | 0.0667 | 741.111** | 2.92 | 3.96 |
| Source x level | 1 | 0.0016 | 0.0016 | 17.778** | 2.92 | 3.96 |
| Control vs others | 1 | 0.0254 | 0.0254 | 282.222** | 2.92 | 3.96 |
| Error (a) | 25 | 0.0023 | 0.00009 |  |  |  |
| Times | 7 | 0.0020 | 0.00028 | 3.630** | 3.19 | 4.09 |
| Times x levels | 28 | 0.0125 | 0.00045 | 5.840** | 3.97 | 4.91 |
| Time x source | 3 | 0.0007 | 0.0002 | 2.590NS | 2.92 | 3.80 |
| Time x level | 3 | 0.0042 | 0.0014 | 18,180** | 2.92 | 3.80 |
| Time x source x level | 3 | 0.0021 | 0.0007 | 9.090** | 2.92 | 3.80 |
| Error (b) | 175 | 0.0136 | 0.000077 |  |  |  |

NS Non significant.

** Highly significant (P<0.01).

Table A-11. ANOVA of selenium concentrations (mg/kg) of bahiagrass sprayed with five levels of a selenium fertilizer (n = 100).

| | | | | | Required F | |
|---|---|---|---|---|---|---|
| Sources of variance | df | Sum of squares | Mean squares | F value | 0.05 | 0.01 |
| Blocks | 3 | 0.02644 | 0.00881 | 1.24 | | |
| Levels | 4 | 0.17917 | 0.04479 | 6.30** | 3.26 | 5.41 |
| Error (a) | 12 | 0.08519 | 0.00710 | | | |
| Times | 4 | 0.22413 | 0.05603 | 7.36** | 2.52 | 3.65 |
| Times x levels | 16 | 0.17681 | 0.01105 | 1.45NS | 1.81 | 2.32 |
| Error (b) | 60 | 0.45715 | 0.00761 | | | |

NS Non significant.
** Highly significant (P<0.01).

Table A-12. ANOVA of selenium concentrations (mg/kg) of fescue topdressed with five levels of a selenium fertilizer (n = 120).

| Sources of variance | df | Sum of squares | Mean squares | F value | Required F 0.05 | Required F 0.01 |
|---|---|---|---|---|---|---|
| Blocks | 3 | 10.56 | 3.52 | | | |
| Levels | 4 | 134.71 | 33.68 | 10.02** | 3.26 | 5.41 |
| Error (a) | 12 | 40.37 | 3.36 | | | |
| Times | 5 | 191.49 | 38.29 | 12.98** | 2.34 | 3.27 |
| Times x levels | 20 | 206.62 | 10.33 | 3.50** | 1.71 | 2.13 |
| Error (b) | 75 | 221.25 | 2.95 | | | |

** Highly significant ($P<0.01$).

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Guillermo Valle was born on September 11, 1948, in La Ceiba , Honduras. He attended grade school and high school in his country, Honduras. After high school, in 1967 he began Animal Husbandry studies at Instituto Tecnologico y de Estudios Superiores in Monterrey, Mexico. He graduated with an Ingeniero Agronomo Zootecnista degree in 1972.

After graduation he returned to Honduras and in March of 1973 started working as an Assistant Professor at the Universidad Nacional Autonoma de Honduras'regional center in La Ceiba., where he taught Poultry Science and Ag Extension from 1973 to 1976. In the fall of 1976 he began studies toward a Master of Science degree at Universidad Nacional de Colombia-Instituto Colombiano Agropecuario joint program of graduate studies at Bogota, Colombia graduating in 1978 with an M.S. in Animal Production.

Upon returning to Universidad Nacional Autonoma de Honduras he became a Professor and taught Dairy Cattle Production and Tropical Pastures as well as conducting research in dairy and dual-purpose cattle nutrition and tropical pastures. He was at the Dairy Science Department of the University of Florida as a visiting professor from August 1990 to August 1992 funded by a Fulbright grant and wrote two books, Milk Production in the Tropics and Tropical Pastures and Forages. He then reassumed his work at Universidad Nacional Autonoma de Honduras, but returned to University of Florida in August 1996 to start graduate studies in the Animal Science Department where he is currently a PhD candidate.

I certify that I have read this study and in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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